A RANDOMIZED DOUBLE BLINDED STUDY OF ULTRASOUND GUIDED TRANSVERSE ABDOMINIS PLANE BLOCK FOR POST OPERATIVE ANALGESIA FOR CESAREAN SECTION WITH BUPIVACAINE VS BUPIVACAINE WITH DEXAMETHASONE (PERINEURAL VS INTRAVENOUS)

A STUDY OF 60 CASES

DISSERTATION

SUBMITTED IN PARTIAL FULFILMENT OF UNIVERSITY

REGULATIONS FOR THE AWARD OF

M.D. DEGREE EXAMINATION

BRANCH X – ANAESTHESIOLOGY

THE TAMIL NADU

Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

MAY, 2018.

CERTIFICATE

This is to certify that this dissertation **"A RANDOMIZED DOUBLE BLINDED STUDY OF ULTRASOUND GUIDED TRANSVERSE ABDOMINIS PLANE BLOCK FOR POST OPERATIVE ANALGESIA AFTER CESAREAN SECTION WITH BUPIVACAINE VS BUPIVACAINE WITH DEXAMETHASONE (PERINEURAL VS INTRAVENOUS)"**presented herein by **Dr. S.RAMYA** is an original work done in the Department of Anaesthesiology, Kanyakumari Govt Medical College Hospital, Asaripallam, Nagercoil for the award of Degree of M.D (Branch – X) Anaesthesiology under my guidance, during the academic period of **2015 – 2018**.

DEAN,

Kanyakumari Govt Medical College Hospital,

Asaripallam, Nagercoil – 629201.

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DECLARATION

I, **Dr. S.RAMYA** hereby declare that the dissertation title **"A RANDOMIZED DOUBLE BLINDED STUDY OF ULTRASOUND GUIDED TRANSVERSE ABDOMINIS PLANE BLOCK FOR POST OPERATIVE ANALGESIA AFTER CESAREAN SECTION WITH BUPIVACAINE VS BUPIVACAINE WITH DEXAMETHASONE (PERINEURAL VS INTRAVENOUS) "**has been done by me. This is submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. degree, Branch – X (ANAESTHESIOLOGY) Degree Examination to be held in **May 2018**.

Place : Asaripallam

Date : DR.S.RAMYA

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URKUND

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Analysed Document: A RANDOMIZED DOUBLE BLINDED STUDY OF ULTRASOUND GUIDED TAP BLOCK FOR POST OPERATIVE ANALGESIA FOR CESAREAN SECTION WITH BUPIVACAINE VS BUPIVACAINE WITH DEXAMETHASONE (PERINEURAL VS INTRAVENOUS).docx (D31657351)
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Sources included in the report:

http://www.usra.ca/regional-anesthesia/specific-blocks/trunk/tfpblock.php

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INTRODUCTION

Pain is the 5th vital sign that has to be monitored in the postoperative period. Pain is defined as a sensory or emotional unpleasant experience associated with actual or related tissue damage. [14]

It is one's fundamental right to be pain free. In women who undergoes lower segment caesarean section the postoperative pain is felt as moderate to severe. It is challenging for the anaesthesiologist to make their patients pain free. Postoperatively, for women undergoing caesarean section, pain has to be addressed effectively with minimal interruptions and complications in order to make these women alert and comfortable to take care of their newborn.

The use of peripheral nerve blockade has grown in popularity because it decreases pain as assessed by visual analogue scores postoperatively, decreases the need for postoperative analgesics, decreases the incidence of nausea, shortens postanesthesia care unit time, and increases patient satisfaction. [2]

Abdominal field blocks have been around for a long time and have been extensively used as they are mostly technically unchallenging. An ultrasound-guided approach of the Transverse abdominis plane block was first described in 2007 by Hebbard et al. The ultrasound technique was induced to improve the success rate of the TAP block. This ultrasound procedure is performed with ultrasound high frequency (5–13 MHz) probe which is placed on the lateral

abdominal wall between the costal margin and the iliac crest at the anterior axillary line. The technique involves injection of local anesthetic solution into a plane between internal oblique muscle and transversus abdominis muscle. This plane contains the thoracolumbar nerves originating from T6 to L1, ilioinguinal and iliohypogastric nerves. They supply sensory blockade to the skin, muscles and parietal peritoneum of the anterolateral abdominal wall. Real-time ultrasonography facilitates easy needle visualization and visualization of the local anesthetic spread in to the target "fascial plane" which is parallel to the ultrasound probe.

There is a new strategy to prolong analgesia beyond the pharmacological duration of the local anesthetics. They include introduction of perineural catheters to allow prolonged infusion of the Local anesthetics or co-administration of adjuvants such as Epinephrine, α2 agonists (i.e. Clonidine), Midazolam or Corticosteroides – Dexamethasone . The perineural catheter techniques can be very effective and can provide analgesia for several days, but this technique is limited with difficulties in placement and removal of the catheter, or rarely, with infection . It is believed that dexamethasone as a supplement improves the quality and duration of the local anesthetics. This is thought to be mediated by attenuating the release of inflammatory mediators, reducing ectopic neuronal discharge, and inhibiting potassium channel-mediated discharge of nociceptive C-fibres .

Transverse abdominis plane block in post-cesarean patients has revolutionised the acute pain management avoiding the side effects of opioids and NSAIDS. Dexamethasone added to local anaesthetics have been proved to prolong the duration of analgesia.

The mechanism of action of dexamethasone on peripheral nerve during perineural injection is unknown.Desmet et al. concluded that systemic (intravenous) injection of Dexamethasone is equivalent to perineural Dexamethasone injection about the prolonging the analgesic duration of single-shot interscalene block with Ropivacaine. Kawanishi et al. have come to an opposite conclusion that perineural but not systemic Dexamethasone prolongs the duration of interscalene block .This study is designed to compare the duration of post operative analgesia provided by ultrasound guided bilateral transverse abdominis plane block with bupivacaine vs bupivacaine with dexamethasone (perineural vs intravenous) for cesarean section.

AIM OF THE STUDY

To compare the duration of Postoperative Analgesia with Bupivacaine Vs Bupivacaine with Dexamethasone (perineurally or intravenously) in patients undergoing caesarean section using Ultrasound guided Transverse Abdominis Plane Block.

ANATOMY OF ANTERIOR ABDOMINAL WALL

THE MUSCLES AND FASCIA OF THE ABDOMEN

THE SUPERFICIAL FASCIA

The superficial fascia of the abdomen consists of a single layer containing a variable amount of fat; but near the groin it is easily divisible into two layers, between which are found the superficial vessels and nerves and the superficial inguinal lymph glands.

SUPERFICIAL LAYER

Fascia of Camper is a thick, areolar in texture, and contains in its meshes a varying quantity of adipose tissue. Below, it passes over the inguinal ligament, and is continuous with the superficial fascia of the thigh.

In the male, Camper's fascia is continued over the penis and outer surface of the spermatic cord to the scrotum, where it helps to form the dartos. As it passes to the scrotum it changes its characteristics, becoming thin, destitute of adipose tissue, and of a pale reddish color, and in the scrotum it acquires some involuntary muscular fibers.

From the scrotum it may be traced backward into continuity with the superficial fascia of the perineum.In the female, Camper's fascia is continued from the abdomen into the labia majora.

DEEP LAYER

Fascia of Scarpa is a thinner and more membranous in character than the superficial, and contains a considerable quantity of yellow elastic fibers. It is loosely connected by areolar tissue to the aponeurosis of the external oblique muscle, but in the middle line it is more intimately adherent to the linea alba and to the symphysis pubis, and is prolonged on to the dorsum of the penis, forming the fundiform ligament; above, it is continuous with the superficial fascia over the rest of the trunk; below and laterally, it blends with the fascia lata of the thigh a little below the inguinal ligament; medially and below, it is continued over the penis and spermatic cord to the scrotum, where it helps to form the dartos. From the scrotum it may be traced backward into continuity with the deep layer of the superficial fascia of the perineum. In the female, it is continued into the labia majora and hence to the fascia of Colles.

EXTERNAL OBLIQUE MUSCLE

The external oblique muscle is the largest and most superficial of the 3 muscles. It runs inferiorly from the external, inferior surfaces of the lower 8 ribs. The fibres originating from the lower ribs run inferiorly and insert into the iliac crest. The fibres originating from the middle and uppermost ribs run infero-anteriorly and end in a thick aponeurosis. Anteriorly, the aponeurosis joins the aponeurosis from the transverse abdominis and internal oblique muscles forming the linea alba. Inferiorly the aponeurosis forms the inguinal ligament.

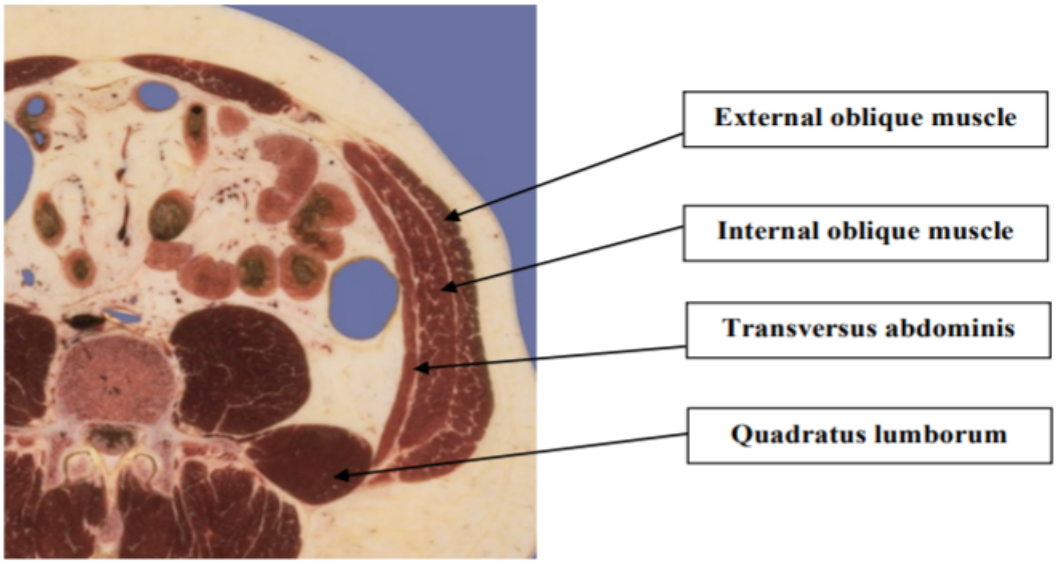
INTERNAL OBLIQUE MUSCLE

The internal oblique muscle is a smaller thinner muscle than the external oblique. It originates from the inguinal ligament and the iliac crest. Its' fibres cover the anterolateral part of the abdomen inserting anteriorly into the linea alba, above the transverse abdominis muscle, and superiorly into the cartilages of the lower 6 ribs.

TRANSVERSE ABDOMINIS MUSCLE

The transverse abdominis muscle is the most internal of the 3 muscle layers, lying directly beneath the internal oblique muscle. Its' fibres arise from the inguinal ligament, the iliac crest, the lumbodorsal fascia and the inner surfaces of the cartilages from the lower 6 ribs.

It's fibres run transversely across the abdomen ending in a broad aponeurosis. This aponeurosis is formed more laterally than the aponeurosis of the external and internal oblique muscles. It continues medially and inserts into the linea alba.



THE LINEA ALBA

The linea alba is a tendinous raphe in the middle line of the abdomen, stretching between the xiphoid process and the symphysis pubis. It is placed between the medial borders of the Recti, and is formed by the blending of the aponeurosis of the Oblique and Transverse. It is narrow below, corresponding to the linear interval existing between the Recti; but broader above, where these muscles diverge from one another. At its lower end the linea alba has a double attachment—its superficial fibers passing in front of the medial heads of the Recti to the symphysis pubis, while its deeper fibers form a triangular lamella, attached behind the Recti to the posterior lip of the crest of the pubis.

LINEA SEMILUNARIS

The linea semilunaris are two curved tendinous lines placed one on either side of the linea alba. Each corresponds with the lateral border of the Rectus,

extends from the cartilage of the ninth rib to the pubic tubercle, and is formed by the aponeurosis of the internal oblique at its line of division to enclose the Rectus, reinforced in front by that of the external oblique, and behind by that of the Transversus.

TRANSVERSALIS FASCIA

The transversalis fascia is a thin aponeurotic membrane which lies between the inner surface of the Transversus and the extraperitoneal fat. It forms part of the general layer of fascia lining the abdominal parietes, and is directly continuous with the iliac and pelvic fascia. In the inguinal region, the transversalis fascia is thick and dense in structure and is joined by fibers from the aponeurosis of the Transversus, but it becomes thin as it ascends to the diaphragm, and blends with the fascia covering the under surface of this muscle.

Behind, it is lost in the fat which covers the posterior surfaces of the kidneys. Below, it has the following attachments: posteriorly, to the whole length of the iliac crest, between the attachments of the Transversus and Iliacus; between the anterior superior iliac spine and the femoral vessels it is connected to the posterior margin of the inguinal ligament, and is there continuous with the iliac fascia. Medial to the femoral vessels it is thin and attached to the pubis and pectineal line, behind the inguinal aponeurotic falx, with which it is united; it descends in front of the femoral vessels to form the anterior wall of the femoral sheath. Beneath the inguinal ligament it is strengthened by a band of fibrous

tissue, which is only loosely connected to the ligament, and is specialized as the deep crural arch.

NERVE SUPPLY OF ANTERIOR ABDOMINAL WALL

Anterolateral abdominal wall receives nerve supply from,

1.Branches from the anterior rami include the intercostal nerves (T7-T11)

2. Subcostal nerve (T12)

3.Iliohypogastric and Ilioinguinal nerves (L1).

As they become more superficial, provide lateral cutaneous and anterior cutaneous branches.

- The T7 to T11 intercostal nerves leave the intercostal space between the internal oblique and the transversus abdominis muscles.
- The subcostal nerve (T12) and the ilioinguinal and iliohypogastric nerves (L1) also lies in between the transversus abdominis and internal oblique, and supply these muscles.
- The Thoracic nerves T7-T12 courses anteriorly to pierce the rectus sheath and continue as anterior cutaneous nerves.
- The thoracic nerves, T7 to T12 gives motor branched to pyramidalis and the rectus muscle and have cutaneous branches laterally in the abdomen.
- Nerves from T7 - T11 provide sensory supply to the skin, costal parts of diaphragm, related parietal pleura and the peritoneum.

- T7 provide sensory innervation at the epigastrium,
- T10 at the umbilicus, and
- L1 at the groin.

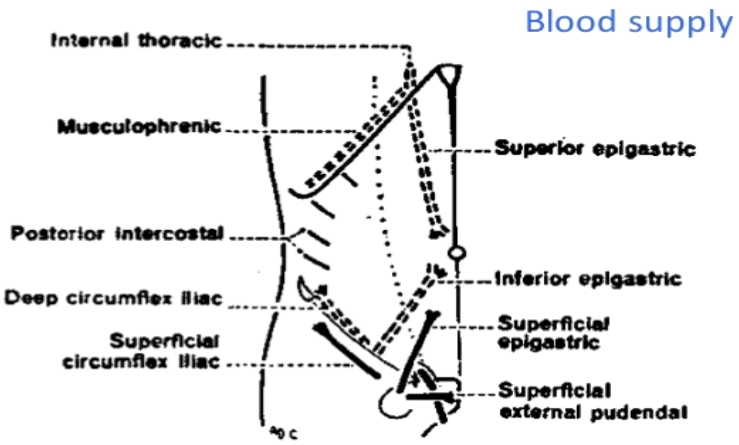
BLOOD SUPPLY TO THE ANTERIOR ABDOMINAL WALL

Three arterial branches provide blood supply to the anterior abdominal wall,[22]

1. **The Deep Inferior Epigastric artery** and vein originate from the external iliac vessels.

2. **The Deep Circumflex Iliac Artery** - branch of the external iliac artery, runs between the Transverse Abdominis Muscle and the Internal oblique muscle parallel to the inguinal ligament.

3.**The Superior Epigastric Artery** -branch of the internal thoracic artery and vein enter the rectus sheath superiorly and anastomose with the inferior epigastric vessels.

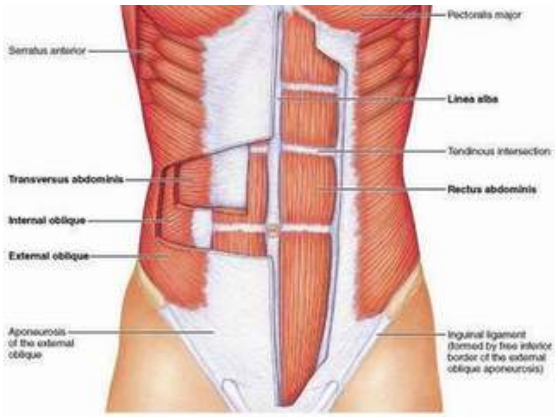


ANATOMY OF TRANSVERSE ABDOMINIS PLANE

The lateral abdominal wall consists of subcutaneous tissue, the external oblique muscle, the internal oblique muscle, and the transversus abdominis muscle progressing from superficial to deep.

The fascial sheath [18]that lies deep to the internal oblique muscle and superficial to the transversus abdominis muscle is the target for the transverse abdominis plane (TAP) block. The nerves that exit the thoracolumbar spinal column pass laterally through the fascial layer to innervate the abdominal wall.

The landmarks for the TAP block are referred to as the *Triangle of Petit*, and is bound anteriorly by the External oblique Muscle, posteriorly by the Latissimus dorsi and inferiorly by the iliac crest. It is located along the midaxillary line inferior to the lower costal margin and superior to the iliac crest.[3]

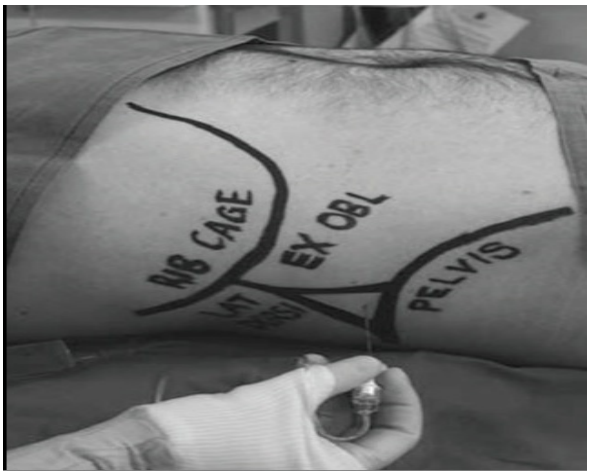


TRANSVERSE ABDOMINIS PLANE BLOCK

TECHNIQUES

1. LOSS OF RESISTANCE (BLIND INJECTION)

The point of entry for the blind TAP block is the lumbar triangle of petit .This technique relies on feeling double “pops” as the needle traverses the external oblique and internal oblique muscles. [18]A blunt needle will make the loss of resistance more significant.

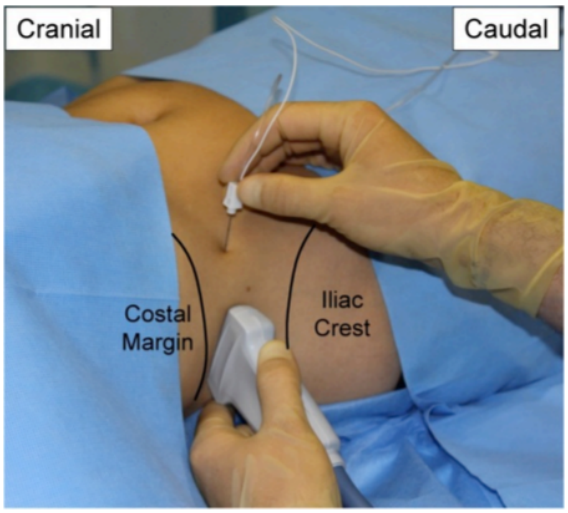


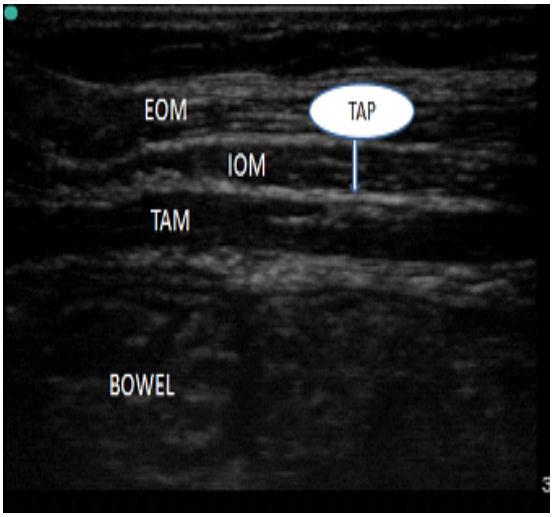
2. ULTRASOUND GUIDED

The patient is positioned supine. Using sterile technique the ultrasound probe is placed firmly superior and parallel to the iliac crest.

A 21-gauge, 10-cm insulted needle is inserted several centimeters medial to the probe using an in-plane approach. [18]Bowel peristalsis can often be seen just deep to the transversus abdominis muscle layer.

After negative aspiration, 15 to 20 mL of local anesthetic is incrementally injected under ultrasound visualization.





SIDE EFFECTS AND COMPLICATION [18]

1.Transient Femoral Nerve Block

2.Peritoneal puncture is possible (even with ultrasound guidance), and there has been one reported case of a liver hematoma following a blind TAP block technique.[4]

3.Hemorrhagic complications – localized bruising and tenderness to severe hematomas

4.Nerve damage

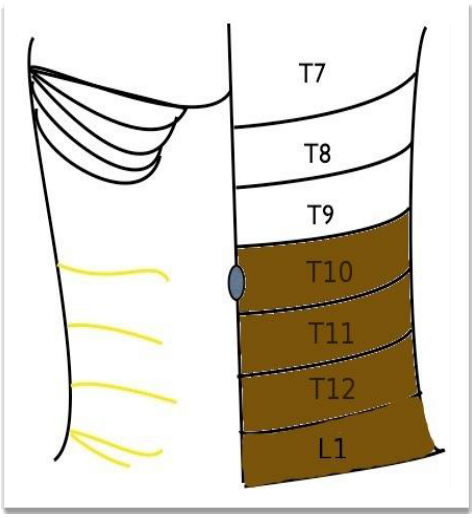
5.Infection

6.Systemic Toxicity

APPLICATIONS

Transverse Abdominis Plane blocks are indicated for any lower abdominal surgery, including

- Hernia repair
- Appendicectomy
- Caesarean delivery
- Abdominal hysterectomy
- Laparoscopic surgery
- Renal transplantation and
- Prostatectomy.
- Bilateral blocks can be used for midline incisions or laparoscopic procedure. It is reasonable to expect analgesia between T10 and L1 with a single injection.



SPREAD

The dermatomeric extent of the Transverse abdominis plane block and its indications are currently under discussion. It is not clear if the local anesthetic blocks somatic nerves alone or if it also spreads to block autonomic nerves.

Radiological Computerized Tomography and Magnetic Resonance Imaging have evidenced the spread of local anesthetic beyond the transverse abdominis plane to the quadratus lumborum and to the intrathoracic paravertebral regions.

The classical Transverse Abdominis Plane block may not reliably provide analgesia for procedures above the level of the umbilicus that is innervated by T10 endings .The extension is generally from L1 to T10 However, a T7 to L1 extension has been also reported [25]

The subcostal Transverse Abdominis Plane block may produce a T9 to 11 block extent in more than 60% of cases . In children, ultrasound-guided supra-iliac Transverse abdominis plane block with 0.2 ml/kg of anesthetic performed by novice operators, produced lower abdominal sensory blockade of only 3 to 4 dermatomes [25]. Only 25% of transverse abdominis plane blocks may have upper abdominal block extension.

Thus, the optimal local anesthetic concentration, the duration of effect and utility of these blocks in children needs clarification. [25]

PRINCIPLES OF ULTRASOUND

Ultrasound is an imaging modality which relies upon the use of sound waves which are transmitted into the body and then reflected back again from the structures being examined.

The device which transmits the sound pulses is called a transducer. This contains a piezo-electric crystal which is able to convert electrical signal into sound and then convert the returning sound wave back into electrical signals again.

Different reflecting surfaces or interfaces within the body reflect sound to different degrees. A strong reflector appears as a bright white area (echogenic) on the image, whereas areas from which no echoes have been obtained appear as black (anechoic).

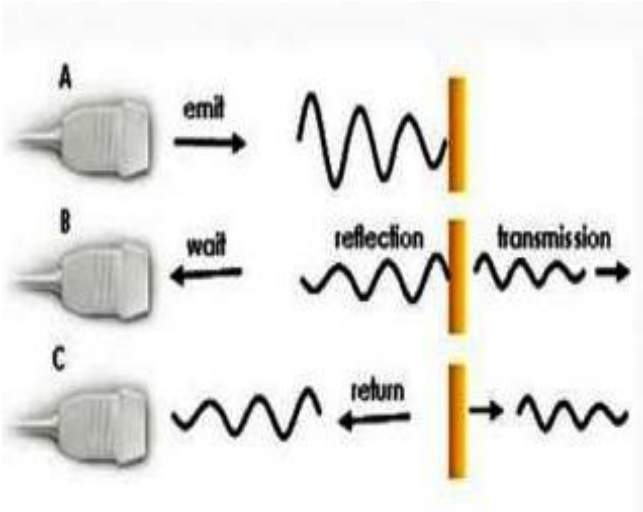
Every ultrasound image is composed of a discrete number of lines of echo data placed side by side to appear continuous. Multiple lines of data are built up as the ultrasound beam sweeps through the field of view.

In order to build up each of the single lines of data, the time taken for the returning echo is measured. This allows the depth of the reflecting interface to be determined. Each sweep of the ultrasound beam produces one frame of data (composed of multiple lines). Many complete sweeps are performed every second which produces the frame rate (frames per second). The operation is analogous to the operation of a television camera. At any one moment, the ultrasound beam is scanning along one of the many lines of sight which will

ultimately form the image. The image is constantly updated at the prevailing frame rate. This can be frozen and hard copy images produced.

The resolution of the picture produced depends on the wavelength of the sound wave used – this can be varied. There is a trade off between resolution and penetration. Higher frequency transducers allow better resolution, but as the frequency is increased penetration is reduced.

As a general rule, the highest frequency transducer should be used to achieve the penetration required. An ultrasound image is a map of reflectivity of the body part scanned. Organs containing multiple interfaces will produce multiple echoes and this is characteristic of solid organs like liver, spleen and kidneys. Structures containing no interfaces will appear echo-free, such as liquid or urine in the bladder.



PIEZOELECTRIC EFFECT

Some materials can produce electric energy in response to a mechanical stress and conversely, produce a mechanical response when an electric current travels through them. This is the so-called piezoelectric effect.

Medical ultrasound waves are produced by a piezoelectric crystal as a consequence of the mechanical response to an electric field. The transducer also picks up the reflected waves or “echoes” from the tissues and converts them into electrical signals that are used to form real-time images on a computer. The crystal thus both transmits and receives the sound.

Each electric signal is registered, amplified, and filtered to reduce noise, and the depth of the tissue that has generated the echo is calculated. The signals are then digitalized and processed in order to produce an image.[24]

- Early ultrasound devices used a single crystal to create a one dimension image, called a-mode image.
    - Modern machines generate a b-mode or two-dimensional or gray-scale image created by 128 or more crystals.
    - Each crystal receives a pulse that produces a scan line used to create an image on the screen.
    - This image is renewed several times each second to produce a real-time image.

IMAGING

Depending on the medium's physical properties and the contact with different interfaces into the medium, the energy of the wave is dissipated, attenuated and reflected. At the interface where one tissue borders and other tissue, the wave is refracted and reflected back as an echo. some structures will completely absorb the sound waves. Thus, echoic tissues are those tissues that reflect the wave whereas anechoic tissues do not reflect the wave.

Ultrasounds penetrate well through fluids that are anechoic and appear as black on the monitor. Fluids allow ultrasounds to pass through more or less attenuated until they encounter the surface of denser structures. Bone or air are poorly penetrated by ultrasounds and generate a kind of "sound-shadow". The transverse appearance of nerves is round or oval and hypo-echoic. They may appear as honeycomb structures containing hyper-echoic points or septa inside them. Nerves are surrounded by a hyper-echoic border that corresponds to connective tissue. Tendons have a similar appearance. On the longitudinal scan, tendons disappear while tracking them for some distances whereas nerves do not disappear.

Blood vessels appear as round hypo-echoic structures with a well defined hyper-echoic border corresponding to the vessel wall. The arteries are not compressible and are pulsating, veins have a thinner border and are compressible

Muscles appear as heterogeneous or homogeneous hypo-echoic structures with hyper-echoic septa and a fibrous-lamellar texture. The periostium appears as hyper-echoic as it reflects entirely the echoes.

As a consequence, the bone underlying the periostium appears as black . Since the speed of the wave in different tissues is known, the time for the reflected wave to return back indicates the depth of the tissue.All this information is converted into a two-dimensional image on the screen.

OUT OF PLANE APPROACH

Out of plane approach is by inserting the needle so that it crosses the plane of imaging near the target.This approach has an advantage of easier to perform , less patient discomfort and short needle insertion path.Also has a disadvantage of difficulty in finding the echogenic dot as the needle crosses the ultrasound beam.

IN PLANE APPROACH

In plane approach is by inserting the needle within the plane of imaging to visualize the entire shaft and tip.This approach has an advantage of ability to track the needle tip.Also has a disadvantage of more time consuming , difficult to perform , and can be more painful due to longer insertion path.

APPLIED PHYSIOLOGY

PAIN

Pain was called by Sherrington, "the physical adjunct of an imperative protective reflex." Painful stimuli initiate withdrawal response , avoidance responses and is associated with an unpleasant affect.[12] It turns out to be immensely complex because when pain is prolonged and tissue is damaged, central nociceptor pathways are sensitized and reorganized.

NOCICEPTORS & THERMORECEPTORS

Pain and temperature sensations arise from unmyelinated dendrites of sensory neurons located around hair follicles throughout the glabrous and hairy skin as well as deep tissue.

Impulses from nociceptors (pain) are transmitted via two fibers.They are myelinated A delta fibers (2–5 mm in diameter) and an unmyelinated C fibers (0.4–1.2 mm in diameter) . Thermoreceptors also span these two fiber types. Cold receptors are on dendritic endings of A delta fibers and C fibers, whereas warmth (heat) receptors are on C fibers.

Pain may be classified as that causes a "bright," sharp, localized sensation (fast pain) followed by a dull, intense, diffuse, and unpleasant feeling (slow

pain).Fast pain is due to activity in the A delta pain fibers, whereas slow pain is due to activity in the C pain fibers.

Pain is also classified as physiologic or acute pain and pathologic or chronic pain, which includes inflammatory pain and neuropathic pain. Acute pain has a sudden onset and recedes during the healing process and it serves an important protective mechanism. Chronic pain can persists long after recovery from an injury and is often refractory to common analgesic agents, including nonsteroidal anti-inflammatory drugs and opiates. Chronic pain can result from nerve injury (neuropathic pain) including diabetic neuropathy, toxin-induced nerve damage, and ischemia.

VISCERAL PAIN

It is a poorly localized, unpleasant, and associated with nausea and autonomic symptoms, visceral pain often radiates or is referred to other areas.The autonomic nervous system, like the somatic, has afferent components, central integrating stations, and effector pathways. The receptors for pain and the other sensory modalities present in the viscera are similar to those in skin, but there are marked differences in their distribution. There are no proprioceptors in the viscera, and few temperature and touch receptors. Nociceptors are present, although they are more sparsely distributed than in somatic structures.

Afferent fibers from visceral structures reach the CNS via sympathetic and parasympathetic nerves. Their cell bodies are located in the dorsal roots and the homologous cranial nerve ganglia. Specifically, there are visceral afferents in the facial, glossopharyngeal, and vagus nerves; in the thoracic and upper lumbar dorsal roots; and in the sacral roots . There may also be visceral afferent fibers from the eye in the trigeminal nerve.

REFERRED PAIN

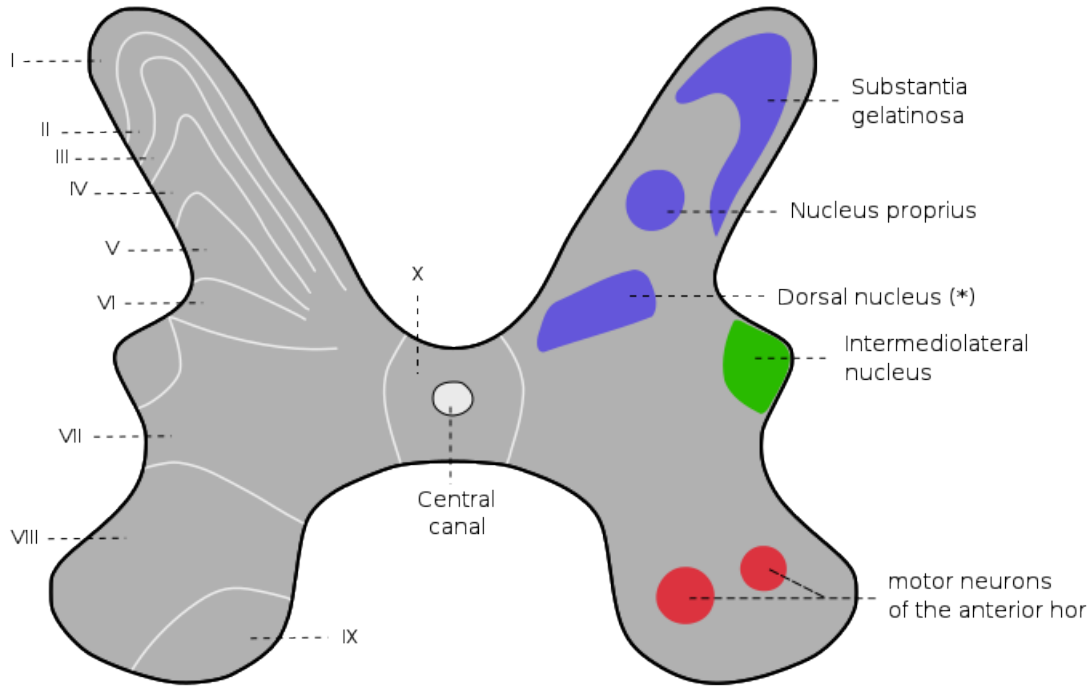
Irritation of a visceral organ produces pain that is felt not at that site but in some somatic structure that may be a considerable distance away. When pain is referred, it is usually to a structure that developed from the same embryonic segment or dermatome as the structure in which the pain originates. This principle is called the dermatomal rule. The basis for referred pain may be convergence of somatic and visceral pain fibers on the same second-order neurons in the dorsal horn that project to the thalamus and then to the somatosensory cortex . This is called the convergence–projection theory.

DORSAL HORN

The dorsal horns are classified based on the histologic characteristics into laminae I–VII, with I being the most superficial and VII the deepest. Lamina VII receives afferents from both sides of the body, whereas the other laminae receive only unilateral input. Lamina II and part of lamina III make up the

substantia gelatinosa.Three types of primary afferent fibers mediate cutaneous sensation:

1. large myelinated A alpha and A beta fibers that transmit impulses generated by mechanical stimuli;
2. Small myelinated A delta fibers, which transmit impulses from cold receptors and nociceptors that mediate pain and some of which transmit impulses from mechanoreceptors; and
3. Small unmyelinated C fibers that are associated with pain and temperature.



DORSAL COLUMN PATHWAY

Fibers mediating pain and temperature sensations ascend ipsilaterally in the dorsal columns to the medulla, where they synapse in the gracilus and cuneate nuclei.

The second-order neurons from these nuclei cross the midline and ascend in the medial lemniscus to end in the contralateral ventral posterior lateral nucleus and related specific sensory relay nuclei of the thalamus. This ascending system is called the dorsal column or medial lemniscal system.

The fibers within the dorsal column pathway are joined in the brain stem by fibers mediating sensation from the head. Touch and proprioception are relayed mostly via the main sensory and mesencephalic nuclei of the trigeminal nerve.

SOMATOTOPIC ORGANIZATION

Within the dorsal columns, fibers from different levels of the cord are somatotopically organized. Specifically, fibers from the sacral cord are positioned medially and those from the cervical cord are lateral. This arrangement continues in the medulla with lower body representation in the gracilus nucleus and upper body representation in cuneate nucleus. The medial lemniscus is organized dorsal to ventral representing from neck to foot.Somatotopic organization continues through the thalamus and cortex.

Ventroposterolateral thalamic neurons carrying sensory information project in a highly specific way to the two somatic sensory areas of the cortex:

1. Somatic sensory area I (SI) -Brodmann's areas 3, 2, and 1 in the postcentral gyrus and
2. Somatic sensory area II (SII) in the wall of the sylvian fissure.

VENTROLATERAL SPINOTHALAMIC TRACT

Fibers from nociceptors and thermoreceptors synapse on neurons in the dorsal horn. A delta fibers terminate primarily on neurons in laminae I and V, whereas the dorsal root C fibers terminate on neurons in laminae I and II. The synaptic transmitter secreted by afferent fibers subserving fast mild pain is glutamate, and the transmitter subserving slow severe pain is substance P.

The axons from these neurons cross the midline and ascend in th ventrolateral quadrant of the spinal cord, where they form the ventrolateral spinothalamic tract . Fibers within this tract synapse in the Ventroposterolateral nucleus of thalamus. Other dorsal horn neurons that receive nociceptive input synapse in the reticular formation of the brain stem (spinoreticular pathway) and then project to the centrolateral nucleus of the thalamus.

Positron emission tomographic and functional magnetic resonance imaging studies in normal humans indicate that pain activates cortical areas SI, SII, and the cingulate gyrus on the side opposite the stimulus. In addition, the

mediofrontal cortex, the insular cortex, and the cerebellum are activated. These technologies were important in distinguishing two components of pain pathways. From Ventroposterolateral nuclei in the thalamus, fibers project to SI and SII. This is the pathway responsible for the discriminative aspect of pain, and is also called the neospinothalamic tract. In contrast, the pathway that includes synapses in the brain stem reticular formation and centrolateral thalamic nucleus projects to the frontal lobe, limbic system, and insula. This pathway mediates the motivational-affect component of pain and is called the paleospinothalamic tract.

In the central nervous system , visceral sensation travels along the same pathways as somatic sensation in the spinothalamic tracts and thalamic radiations, and the cortical receiving areas for visceral sensation are intermixed with the somatic receiving areas.

NEUROBIOLOGY OF PAIN [20]

The experience of pain involves a series of complex neurophysiologic processes that reflect four distinct components:

1.TRANSDUCTION - noxious stimulus is converted to an electrical impulse in sensory nerve endings.

2.TRANSMISSION - conduction of the electrical impulses to the CNS with the major connections for these nerves being in the dorsal horn of the spinal cord

and thalamus with projections to the cingulate, insular and somatosensory cortex.

3.MODULATION - process of altering pain transmission. It is likely that both inhibitory and excitatory mechanisms modulate pain (nociceptive) impulse transmission in the PNS and CNS.

4.PERCEPTION - occur at the thalamus.

CHEMICAL MEDIATORS OF TRANSDUCTION AND TRANSMISSION [3]

SUBSTANCE	SOURCE	EFFECT
BRADYKININ	Macrophages,Plasma kininogen	Activates nociceptors
SEROTONIN	Platelets	Activates nociceptors
HISTAMINE	Platelets,Mast cells	Produces vasodilation,edema,and pruritis potentiates the response of nociceptors to bradykinin
PROSTAGLANDIN	Tissue injury and Cyclo oxygenase pathway	Sensitizes nociceptors
LEUKOTRIENE	Tissue injury and	Sensitizes nociceptors

	Cyclo oxygenase pathway	
EXCESS HYDROGEN IONS	Tissue injury and Ischemia	Increases pain and hyperalgesia associated with inflammation
CYTOKINES (Interleukins, Tumour Necrosis Factor)	Macrophages	Excite and sensitize nociceptors
ADENOSINE	Tissue injury	Pain and hyperalgesia
NEUROTRANSMITTER (Glutamate,Substance - P)	Antidromic release by peripheral nerve terminals after tissue injury	Substance P activates macrophages and mast cells. Glutamate activates nociceptors
NERVE GROWTH FACTOR	Macrophages	Stimulates mast cells to release histamine and serotonin. Induces heat hyperalgesia. Sensitizes nociceptors.

COMPONENTS OF PAIN

Pain has a sensory component including the conscious perception of site, duration and intensity of pain; a motor component (e.g., defensive posture and

withdrawal reflex, an autonomic component (e.g., tachycardia), and an affective component (e.g., aversion). [3]

GATE THEORY

The gate control theory was first proposed by Ronald Melzack and Patrick wall. According to this theory, [20] painful information is projected to the supraspinal brain regions if the gate is open,whereas painful stimulus is not felt if gate is closed by the stimulataneous inhibitory impulses. Usually, rubbing the skin activates large diameter A beta fibers, which are faster than A delta and C fibers conveying painful stimulus.These A beta fibers deliver information about pressure and touch to the dorsal horn and override some of the pain messages (closes the gate) carried by the A delta and C fibers by activating the inhibitory interneurons in the dorsal horn.

PHYSIOLOGICAL CHANGES IN RESPONSE TO TISSUE DAMAGE

Tissue damage, whether from trauma or surgery, initiates changes in the pain pathway that maintain and even increase the pain experienced by the individual. These changes occur at the site of tissue damage (peripheral sensitization) and the dorsal horn of the spinal cord (central sensitization).

It is important to remember that these mechanisms are normal physiological events and serve to encourage the individual to protect the injury from further damage.Both peripheral and central sensitization contribute towards hyperalgesia and allodynia. Hyperalgesia is increased pain in response to a

painful stimulus, and allodynia is pain following a previously non-painful stimulus.

PERIPHERAL SENSITIZATION

THE TRIPLE RESPONSE

Minor injury produces the familiar 'triple response' of redness (capillary dilatation), wheal (oedema) and flare (arteriolar dilatation).Histamine is one of the first substances to be released into the surrounding tissue and is responsible for the wheal. Antidromic conduction in the C neurone releases substance P from nerve endings in the skin, which in turn causes arteriolar dilatation.

THE INFLAMMATORY SOUP

Depending on the magnitude of the injury, additional inflammatory mediators are released, including: Bradykinin, formed from the precursor kallidin – bradykinin sensitizes nociceptors and induces them to release excitatory peptides, including substanceP,Monoamines: serotonin (5HT) and norepinephrine – norepinephrine is released from sympathetic nerve terminals at the site of injury and prostaglandins and leukotrienes, formed from arachidonic acid, which itself is produced following activation of phospholipase A2 by bradykinin.The effect on the C-afferent terminal of this 'inflammatory soup' is one of sensitization . Now, the magnitude of the stimulus required to generate impulses in the C fibre is greatly reduced. Put another way, the threshold for stimulation is lowered.

A stimulus of insufficient intensity to activate nociceptors prior to injury may do so following sensitization.Not only are nociceptors sensitized following injury, but the number of nociceptors is increased. This is a reflection of the fact that perhaps one-third to one-half of the population of nociceptors are in a 'dormant' state and are not stimulated unless tissue damage occurs.

CENTRAL SENSITIZATION

The synapse between the C-afferent neuron and the dorsal horn cell is not simply a relay site of information from one cell to another. It allows modulation of the afferent input, so that the activity in the dorsal horn cell may be increased or decreased depending on the activity of other systems acting on the synapse. Sensitization in the dorsal horn results in an exaggerated response in the dorsal horn cell not only to C-fibre input, but also for A beta input.

WIND-UP

Sensitization increases the dorsal horn cell output for a given C-fibre input. In addition, within a narrow frequency range of Cfibre input, animal studies have demonstrated that WDR neurones respond to a repetitive C-fibre stimulation at a fixed frequency, not with a fixed frequency output, but rather by producing an output whose frequency progressively increases. This frequency-dependent, exaggerated response is termed 'wind-up'.

MECHANISMS OF CENTRAL SENSITIZATION

Wind-up is caused by NMDA receptor activation, and experimentally induced wind-up may be inhibited by NMDA receptor antagonists, such as

ketamine. On the other hand, it appears that A beta sensitization is mediated via inhibition of pathways that use GABA(gamma-aminobutyric acid). GABA is a major inhibitory neurotransmitter in the CNS, and NMDA is an excitatory transmitter. A reduction in activity in inhibitory pathways produces an indirect increase of activity in excitatory pathways. Drugs acting at these receptors may be a potentially useful site for treating pain.

PRE EMPTIVE ANALGESIA

The importance of peripheral and central sensitization in amplifying pain perception has directed research towards preventing these processes. Experimentally, it has been shown that nociceptive stimulation from the periphery causes functional changes in the spinal cord which lead to enhancement and prolongation of the sensation of pain. It has also been shown that prior administration of analgesics may inhibit the development of the hyperexcitability within the spinal cord.

ENDOGENOUS PAIN SUPPRESSION PATHWAYS

There is interindividual variation in the response to pain which in part reflects the capability of the brain to suppress input of pain signals to the nervous system by activating endogenous pain suppression pathways . Three major components are ,[20]

1.Periaqueductal gray and periventricular areas surrounding the aqueduct of Sylvius and portions of the third and fourth ventricles

- transmit signals to next component of endogenous pain suppression system.

2.Raphe magnus nucleus (lower pons and upper medulla) and nucleus reticularis paragigantocellularis (medulla) -transmit signals to next component of the endogenous pain suppression system.

3.Dorsal horns of the spinal cord -analgesia signals can be blocked at this area before being relayed to brain.

Electrical stimulation either in the periaqueductal gray area or in the raphe magnus nucleus can almost completely suppress pain signals entering by way of the dorsal spinal roots. Neurotransmitters, especially enkephalins and serotonin are involved in the endogenous pain suppression system. Enkephalins are believed to cause presynaptic inhibition and postsynaptic inhibition of incoming type C and type A-delta pain fibers where they synapse in the dorsal horns.

Presynaptic inhibition is achieved by blocking calcium ion channels and calcium ions cause the release of neurotransmitters at the synapses. Descending pain pathways are activated as a result of pain detection, discrimination, and perception at the higher levels of the CNS.

Inhibitory and excitatory neuroanatomic pathways originate in the brainstem and descend in the dorsal longitudinal fasciculus to modulate nociception and pain perception . When activated these pathways inhibit nociceptive stimuli.

Neurotransmitters identified in descending pathways include norepinephrine, serotonin, and endogenous opioids. Inhibitory pathways are activated by opioids and excitatory pathways are inhibited by opioids thus providing the mechanism of analgesia induced by drugs such as morphine also tricyclic antidepressants and alpha2-adrenergic agonists. Tricyclic antidepressants block the presynaptic reuptake of serotonin and norepinephrine and thus augment their postsynaptic actions in descending pain suppression pathways.

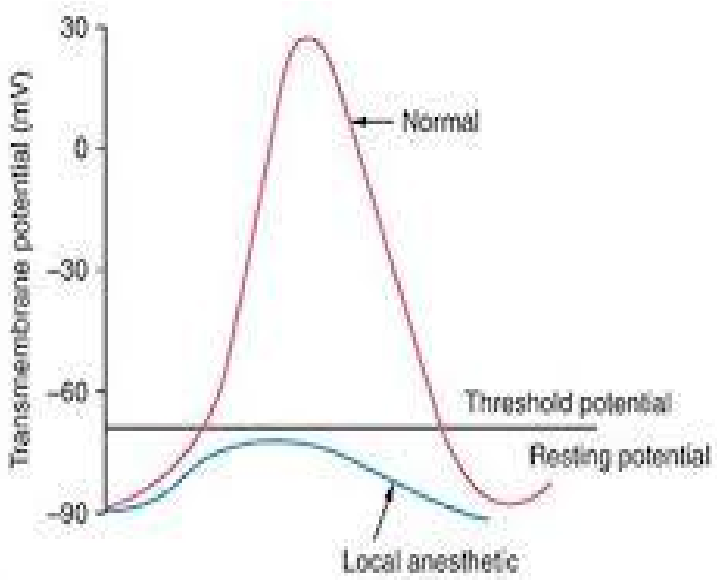
CONSEQUENCES OF POORLY MANAGED ACUTE PAIN [3]

In cardiovascular system , it causes tachycardia,hypertension and increased cardiac work load.In Respiratory system it causes Respiratory muscle spasm ,Decreased vital capacity ,Atelectasis ,Arterial hypoxemia ,Increased risk of pulmonary infection.In Gastrointestinal system it causes postoperative paralytic ileus.In Renal system it causes oliguria and increased chances of urinary infection.It impairs immune system and there is increased chance of thromboembolism.it is also associated with anxiety,muscle fatique and poor patient co operation.

MECHANISM OF ACTION OF LOCAL ANAESTHETICS

Local anesthetics produces conduction blockade by inhibiting the entry of sodium ions through ion-selective sodium channels in nerve membranes. Failure of sodium ion channel permeability to increase, slows the rate of depolarization such that threshold potential is not attained and thus an action potential is not generated.[8]

Blockade of potassium ion channels with much less affinity might explain broadening of the action potential in the presence of local anaesthetics.



Nerve membrane is made up of a bimolecular framework of phospholipid & protein channels protruding through the lipid bilayer. The globular protein is a sodium or potassium ion channel. When the nerve is stimulated, following changes occurs in the cell membrane.[7]

RESTING MEMBRANE POTENTIAL

The neural membrane constantly maintains a voltage difference at rest of –60 to –90 mV[20] between the intracellular medium and the cell's outside, because during rest it is relatively impermeable to sodium ions but selectively permeable Potassium.

DEPOLARIZATION PHASE

During excitation, the Na+ channels open faster and the inwardly directed Na+ current depolarizes the membrane further, thereby leading to opening of more Na+ channels.

REPOLARIZATION PHASE

Upon opening of voltage gated K+ channels, potassium ions pass out of the cell so that electrical neutrality is maintained.

This local ionic current passes relatively uniformly across the nonmyelinated axon, but it is restricted in the myelinated axon, at the nodes of Ranvier.

SODIUM CHANNELS

The sodium channel consists of the large conducting pore (alpha-subunit) and numbers of smaller beta subunits. The alpha-subunit is divided into four subunits (D l- IV). H is the alpha subunit that allows ion conduction and binds to local anesthetics. However, beta subunits may modulate local anesthetic binding to the alpha subunit.

Local anesthetics affinity to the sodium ion channels binding are stereospecific and based on the conformational state of the sodium channel .

During various stages of the action potential Sodium channels exist in ,

1.Activated-open,

2.Inactivated-closed, and

3.Rested closed states.

In the resting nerve membrane, there is equilibrium between the rested-closed and inactivated- closed states sodium channel.

By selectively binding to inactivated-closed states sodium channels, local anesthetic molecules stabilize these channels and prevent their conformational changes to the rested-closed and activated-open states in response to nerve impulses. Sodium channels in the inactivated- closed state are not permeable to sodium, and thus conduction of nerve impulses in the form of propagated action potentials cannot occur.

It is speculated that local anesthetics bind to specific sites located on the inner portion of sodium channels (internal gate or H gate) as well as obstructing sodium channels near their external openings to maintain these channels in inactivated-closed states.

This binding appears to be weak and to reflect a relatively poor fit of the local anesthetic molecule with the receptor. This is consistent with the broad variety of chemical structures that exhibit local anesthetic activity on sodium channels.

FREQUENCY-DEPENDENT BLOCKADE

Sodium ion channels tend to recover from local anesthetic induced conduction blockade between action potentials and to develop additional conduction blockade each time sodium channels open during an action potential (frequency-dependent blockade). Therefore, local anesthetic molecules can gain access to receptors only when sodium channels are in activated-open states.

For this reason, selective conduction blockade of nerve fibers by local anesthetics may be related to the nerve's characteristic frequencies of activity as well as to its anatomic properties, such as diameter. Indeed, a resting nerve is less sensitive to local anesthetic induced conduction blockade than is a nerve that has been repetitively stimulated.

OTHER SITE OF ACTION TARGETS

In addition to sodium ion channels, local anesthetics block voltage-dependent potassium ion channels. Compared with sodium ion channels, local anesthetics exhibit a much lower affinity. However, blockade of potassium ion channels might explain broadening of the action potential in the presence of local anesthetics. Considering the structural similarity between voltage-dependent calcium ion channels and sodium ion channels, it is not surprising that calcium ion currents (L-type most sensitive) may also be blocked by local anesthetics .

Although local anesthetics are considered principally ion channel blockers, there is evidence these drugs may also act on G-protein coupled receptors.

MINIMUM CONCENTRATION

The minimum concentration of local anesthetic necessary to produce conduction blockade of nerve impulses is termed the Cm^{20}.The Cm is similar to the minimum alveolar concentration (MAC) for inhaled anesthetics.

Nerve fiber diameter influences minimum concentration, with larger nerve fibers requiring higher concentrations of local anesthetic for production of conduction blockade. An increased tissue pH or high frequency of nerve stimulation decreases minimum concentration. Each local anesthetic has a unique Cm, reflecting differing potencies of each drug.

The minimum concentration of motor fibers is approximately twice that of sensory fibers; thus, sensory anesthesia may not always be accompanied by skeletal muscle paralysis. Despite an unchanged minimum concentration , less local anesthetic is needed for subarachnoid anesthesia than for epidural anesthesia, reflecting greater access of local anesthetics to unprotected nerves in the subarachnoid space.

Peripheral nerves are comprised of myelinated A and B fibers and unmyelinated C fibers. A minimal length of myelinated nerve fiber must be exposed to an adequate concentration of local anesthetic for conduction blockade of nerve impulses to occur. Both types of pain-conducting fibers (myelinated A-delta and nonmyelinated C fibers) are blocked by similar

concentrations of local anesthetics, despite the differences in the diameters of these fibers. Preganglionic B fibers even though myelinated are more readily blocked by local anesthetics than any fiber.

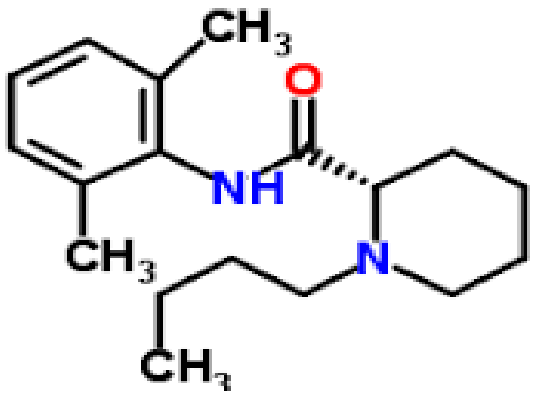
DIFFERENTIAL CONDUCTION BLOCKADE

Selective blockade of preganglionic sympathetic nervous system B fibers using low concentrations of local anesthetics compared to slightly higher concentrations of local anaesthetics interrupt conduction in small C fibers and A fibers.Touch, proprioception, and motor function are still present such that the patient will sense pressure but not pain with surgical stimulation.

CHANGES DURING PREGNANCY

Rapid onset of conduction blockade by local anaesthetics due to increased sensitivity may be present during pregnancy . Alterations in protein-binding characteristics of Bupivacaine may result in increased concentrations of pharmacologically active unbound drug in the parturient's plasma . Progesterone, which binds to the same alpha l-acid glycoprotein as bupivacaine, does not influence protein binding of this local anesthetic .This evidence suggests that Bupivacaine and Progesterone bind to discrete but separate sites on protein molecules.

PHARMACOLOGY OF BUPIVACAINE



Bupivacaine hydrochloride is an amino-amide local anaesthetic synthesized by EKENSTAM et al in 1957 and first used by L.J. Telivuo in 1963. It is available as racemic mixtures of the R & S enantiomers (50:50).

PHYSICOCHEMICAL PROPERTIES

Pk	8.1
% Ionized at PH 7.4	83
Partition Coefficient (Lipid Solubility)	3420
% Protein binding	95

PHARMACOKINETICS

Volume of distribution (l/Kg)	1.02
Clearance (l/Kg/hr)	0.41
T ½ (hour)	3.5

METABOLISM & CLEARANCE

- Metabolism prinicipally by microsomal enzymes located in the liver. Pathways for metabolism of bupivacaine include aromatic hydroxylation, N-dealkylation, amide hydrolysis, and conjugation.
- Bupivacaine undergo renal excretion.

PHARMACODYNAMICS

EFFECT ON CARDIOVASCULAR SYSTEM

Bupivacaine produces dose related effect on the heart.

- In purkinje fibres and ventricular muscles - depresses the rapid phase of depolarization and
- Bupivacaine has high arrhythmogenic potential because of incomplete restoration of sodium channel availability in between action potentials.

EFFECT ON RESPIRATORY SYSTEM

When bupivacaine is systemically absorbed it stimulates the ventilator response to carbon dioxide.

EFFECT ON GASTRO INTESTINAL SYSTEM

When bupivacaine is given as epidural infusion either continuously or intermittently is associated with increased plasma concentrations of transaminases enzyme.

DRUG DOSAGE

The maximal dose of bupivacaine is 2 - 2.5mg/kg. [18]

Strength - 0.125% - 0.75% with or without epinephrine.

CLINICAL USES

1.Spinal anaesthesia

2. Epidural & Caudal anaesthesia

3.Continuous epidural anaesthesia

4. Peripheral nerve block

5. Infiltration anaesthesia

SIDE EFFECTS

1.Allergic reactions

2.Systemic toxicity due to excessive plasma and tissue concentrations.

LOCAL ANAESTHETIC SYSTEMIC TOXICITY [20]

Local anaesthetic systemic toxicity (LAST) range from mild systemic symptoms to cardiovascular symptoms like hypertension, hypotension, tachycardia, bradycardia , ventricular arrhythmia leading to cardiac arrest and central nervous system symptoms like perioral numbness,seizure, respiratory depression,coma and death.

Magnitude of the problem depends on ,

1.Dose administered into the tissues

2. Vascularity of the injection site

3. Presence of epinephrine in the solution

4. Physicochemical properties of the drug.

TREATMENT

A. If signs and symptoms of LAST occur, prompt and effective airway management is crucial to preventing hypoxia and acidosis, which are known to potentiate LAST.

B. If seizures occur, they should be rapidly halted with benzodiazepines. If benzodiazepines are not readily available, small doses of propofol or thiopental are acceptable.

- Although propofol can stop seizures, large doses further depress cardiac function; propofol should be avoided when there are signs of cardiovascular compromise. If seizures persist despite benzodiazepines, small doses of succinylcholine or similar neuromuscular blocker should be considered to minimize acidosis and hypoxemia.
- If cardiac arrest occurs, we recommend standard Advanced Cardiac Life Support with the following modifications:
- If epinephrine is used, small initial doses (10—100 ug boluses in the adult) are preferred.
- Vasopressin is not recommended.

- Avoid calcium channel blockers and ß-adrenergic receptor blockers.
- If ventricular arrhythmias develop, amiodarone is preferred.

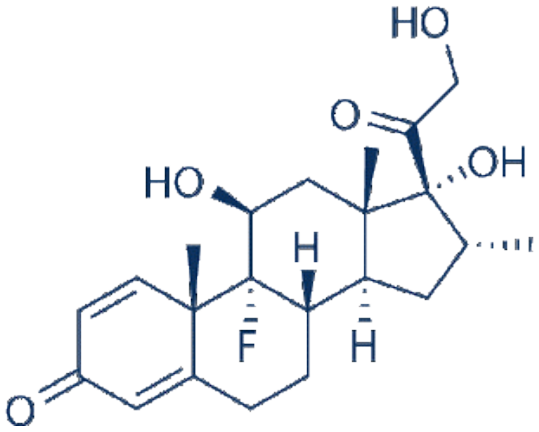
C. Lipid emulsion therapy –

- Consider administering at the first signs of LAST, after airway management

Dosing: 1.5 mL/kg 20% lipid emulsion bolus and 0.25 mL/kg per minute of infusion, continued for at least 10 minutes after circulatory stability is attained

- If circulatory stability is not attained, consider rebolus and increasing infusion to 0.5 mL/kg per minute.
- Approximately 10 mL/kg lipid emulsion for 30 minutes is recommended as the upper limit for initial dosing
- Propofol should not be used as a substitute for lipid emulsion.
- Failure to respond to lipid emulsion and vasopressor therapy should prompt institution of cardiopulmonary bypass (CPB). Because there can be considerable lag in beginning CPB, it is reasonable to notify the closest facility capable of providing it when cardiovascular compromise is first identified during an episode of LAST.

PHARMACOLOGY OF DEXAMETHASONE



Dexamethasone , a synthetic glucocorticoids is a fluorinated derivative of prednisolone and an isomer of betamethasone. The anti-inflammatory effect of 0.75 mg is equivalent to that of 20 mg of Cortisol.

Elimination half life is 3.5 – 5 hours. [18]

Duration of action is 36 – 54 hrs.[18]

MECHANISM OF ACTION [18]

- Acts by Controlling rate of protein synthesis.
- React with cytoplasmic receptors to form a complex which directly influences the rate of RNA transcription
- Synthesis of lipocortins

EFFECTS ON CARDIOVASCULAR SYSTEM

- Positive effect on myocardial contractility
- Causes vasoconstriction by increasing the number of alpha-1-adrenoreceptor and beta-adrenoreceptor
- Prevent edema formation

EFFECTS ON CENTRAL NERVOUS SYSTEM

- Increases the Excitability of the central nervous system.
- In the CSF, Reduction of the release of cytokines (interleukin-1 beta, Tumour necrosis factor).

EFFECTS ON GASTROINTESTINAL SYSTEM

- Increases the likelihood of peptic ulcer disease
- Decreases the gastrointestinal absorption of calcium

EFFECTS ON GENITOURINARY SYSTEM

- Urinary excretion of calcium is increased
- Increases the glomerular filteration rate and stimulates tubular secretory effect
- Has only minor mineralocorticoid activities.

METABOLIC EFFECT

- Stimulates gluconeogenesis and inhibit peripheral utilization of glucose
- Enhance lipolysis
- Reduce conversion of aminoacid to protein

ANTI-INFLAMMATORY EFFECT

Inhibit all stages of the inflammatory process by inhibiting neutrophil and macrophage recruitment, blocking the effect of lymphokines and inhibiting the formation of plasminogen activator

OTHER- It inhibits pituitary corticotrophin release and decrease the output of endogenous corticosteroids.

METABOLISM

Dexamethasone undergoes metabolism in the liver and excreted slowly in the urine, largely as unconjugated steroids.

USES

1.As replacement therapy in congenital adrenocortical deficiency states

2.Allergic disorders

3.Asthma

4.Prevention of post operative and chemotherapy induced nausea and vomiting

5.Cerebral edema

6.Bacterial meningitis – prevents hearing loss

7.Tests for cushing's syndrome

8.Antenatal use in preterm labour

9.Myasthenia gravis

10.Immunosuppression after organ transplantation

REVIEW OF LITERATURE

Several studies were conducted using Dexamethasone as adjuvant to Local anaesthetics for prolongation of post-operative analgesia.

AMANY et al IN 2016 [21] did a study on effect of adding Dexamethasone to Bupivacaine on transversus abdominis plane block for Abdominal Hysterectomy.It was a prospective randomized controlled study ,sixty patients were allocated to receive Transverse abdominis plane block using 20 mL of bupivacaine hydrochloride 0.25% + 2 mL saline 0.9% (control group, *n*=30) and 20 mL of bupivacaine hydrochloride 0.25% + 2 mL dexamethasone “8 mg” (*n*=30).

The primary outcome was postoperative pain, as evaluated by visual analog scale (VAS) for pain scoring at 1, 2, 4, 12, 24 and 48 h postoperatively, whereas the secondary outcomes were time to first analgesia , morphine consumption and the occurrence of nausea, vomiting or somnolence.

The pain score was significantly lower at the postoperative 2 h (4.9 vs. 28.1, *P*=0.01), 4 h (12.2 vs. 31.1, *P*=0.01) and 12 h (15.7 vs. 25.4, *P*=0.02). Furthermore, Time to first analgesia was significantly longer in the dexamethasone group (459.8 vs. 325.4 min, *P*=0.002), with lesser morphine requirements in the postoperative 48 h (4.9 vs. 21.2 mg, *P*=0.003) and lower incidence of nausea and vomiting (6 vs. 14, *P*=0.03).They concluded that

addition of Dexamethasone to Bupivacaine in TAP block prolonged the duration of the block and decreased the incidence of nausea and vomiting.

A Double blinded trial on Effect of Dexamethasone on the duration of interscalene nerve blocks with Ropivacaine or Bupivacaine was conducted by **K.C.CUMMINGS et al IN 2011** [6].Patients were randomized to one of four groups: (i) Ropivacaine: 0.5% Ropivacaine; (ii) Bupivacaine: 0.5% Bupivacaine; (iii) Ropivacaine and steroid: 0.5% Ropivacaine mixed with Dexamethasone 8 mg; and (iv) Bupivacaine and steroid: 0.5% Bupivacaine mixed with Dexamethasone 8 mg.

The primary outcome was considered as time to first analgesic request after post-anaesthesia care unit discharge. Dexamethasone prolonged the duration of analgesia of Ropivacaine Dexamethasone group (22.2 hr) compared to Ropivacaine group (11.8 hr) with a $P < 0.001$ and similarly prolonged the duration of analgesia of Bupivacaine Dexamethasone group (14.8) compared to plain Bupivacaine group (22.4 hr) with a P 0.001]. This study concluded that Dexamethasone prolonged analgesia more with Ropivacaine than Bupivacaine.The combination of Dexamethasone with the local anesthetic provided nearly the same duration of analgesia of twenty-two hours

IN 2006 , MOVAFEGH et al [19]– Conducted a study to assess the prolongation of duration of analgesia using Dexamethasone added to Lignocaine in axillary nerve block.

Sixty patients scheduled for elective hand and forearm surgery under axillary brachial plexus block were randomly allocated to receive either 34 mL Lidocaine 1.5% with 2 mL of isotonic saline chloride (control group, n = 30) or 34 mL lidocaine 1.5% with 2 mL of dexamethasone (8 mg) (dexamethasone group, n = 30) .

After performance of the block, sensory and motor blockade of radial, median, musculocutaneous, and ulnar nerves were recorded at 5, 15, and 30 min The onset time of the sensory and motor blockade was defined as the time between last injection and the total abolition of the pinprick response and complete paralysis.

The duration of sensory and motor blocks were considered as the time interval between the administration of the local anesthetic and the first postoperative pain and complete recovery of motor functions. .

It was summarized that there was no difference in the onset of sensory and motor block between the two groups significantly. The difference in the duration of sensory blockade between Lignocaine with Dexamethasone Vs plain Lignocaine group was 134 minutes. Similarly, the difference in the duration of motor blockade between the two groups was 180 minutes.

It was concluded that blockade was longer in the dexamethasone group than in the lignocaine group with the P value <0.01 which was statistically significant.

IN 2013, DESMET et al[8]-Performed a study on intravenous dexamethasone and its equivalency to perineural dexamethasone in prolonging the analgesic duration of a single-shot interscalene block with Ropivacaine. Patients presenting for arthroscopic shoulder surgery with an Interscalene Block were randomized into three groups: Ropivacaine 0.5% ; Ropivacaine 0.5% and Dexamethasone 10 mg ; and Ropivacaine 0.5% with i.v. Dexamethasone 10 mg.

The primary outcome was the duration of analgesia, defined as the time between performance of the block and the first analgesic request. The median time of a sensory block was equivalent for perineural and i.v. dexamethasone: 1405 min and 1275 min respectively. There was a significant difference between the Ropivacaine group (757 min) and the Dexamethasone groups (P<0.0001) .It was concluded from this study that intravenous Dexamethasone is equivalent to perineural Dexamethasone in prolonging analgesia. Intravenous Dexamethasone at a dose of 0.1- 0.2 killograms could have a comparable analgesic effect and eliminate the need for perineural Dexamethasone injection.

A study to determine the effects of intravenous and perineural dexamethasone on the duration of interscalene brachial plexus block with Ropivacaine in patients undergoing arthroscopic shoulder surgery was done by

KAWANISHI et al IN 2014 [16]. In this prospective, randomized, placebo-controlled trial, patients presenting for arthroscopic shoulder surgery with an Interscalene Block were randomized to receive Ropivacaine 0.75% , Ropivacaine 0.75% plus perineural Dexamethasone 4 mg , or Ropivacaine 0.75% plus intravenous Dexamethasone 4 mg . The primary outcome was the duration of analgesia, defined as the time between performance of the block and the first request for analgesic.The median times of sensory block in Ropivacaine group was 11.2 hours , in perineural Dexamethasone group was 18.0 hours, and in intravenous Dexamethasone group was 14.0 hours

This study demonstrates that perineural but not intravenous administration of Dexamethasone at a dose of 4 mg significantly prolongs the duration of effective postoperative analgesia resulting from a single-shot Interscalene Block with Ropivacaine 0.75%.

DE OLIVEIRA, GS et al IN 2014 [7]- A systematic search was performed to identify randomized controlled trials that evaluated the effects of perineural Dexamethasone as a block adjunct on postoperative pain outcomes in patients receiving regional anesthesia. Meta-analysis was performed using a random-effect model.

Nine randomized trials with 760 subjects were included. The overall effect of seven studies (nine comparisons) that examined perineural Dexamethasone on analgesia duration compared to control favored Dexamethasone with a

weighted mean difference (99%) of 473 minutes . Two studies contained two independent comparisons that were included in the analysis.

Perineural Dexamethasone improves postoperative pain outcomes when given as an adjunct to brachial plexus blocks and there were no reports of persistent nerve injury attributed to perineural administration of the drug.

Animal studies that have been performed by **BAILARD, ORTIZ, FLORES et al IN 2014** [2] have not shown long-term changes in the nerve structure or function after local steroid administration. Studies have shown that nerve injury is a rare complication of dexamethasone injection. Injury that occurs is most often caused by needle trauma compared to the medication that is injected.

A study was done by **BEENA et all in 2013** [4] to assess the analgesic efficacy of ultrasound guided transverse abdominis plane block for retroperitoneoscopic donor nephrectomy. In this prospective randomized double-blind study, 60 patients undergoing laparoscopic donor nephrectomy were randomly divided into two groups by closed envelope method. At the end of surgery, ultrasound guided transverse abdominis plane block was given to the patients of both the groups.

Study group (group S) received inj. Bupivacaine (0.375%), whereas control group (group C) received normal saline. Inj. Tramadol (1 mg/kg) was given as rescue analgesic at visual analog scale more than 3 in any group at rest or on

movement. The analgesic efficacy was assesed by visual analog score both at rest and on movement, time to first dose of rescue analgesic, cumulative dose of tramadol, sedation score, and nausea score, which were also noted at 30 min, 2, 4, 6, 12, 18, and 24 h postoperatively. Total tramadol consumption at 24 h was also assessed.

This concluded that patients in group S had significantly lower Visual analog score, longer time to first dose of rescue analgesic (547.13±266.96 min vs. 49.17±24.95 min) and lower tramadol consumption (103.8±32.18 mg vs. 235.8±47.5 mg) in 24 hours.

MCDERMOTT et al IN 2012 [18] **–** conducted a study to evaluate the placement of the needle tip and local anaesthetic during transversus abdominis plane blocks using the landmark-based ‘double-pop’ technique.In this prospective randomized study,after induction of general anaesthesia, 36 adult patients had a transverse abdominis plane block performed bilaterally using the standard landmark-based technique. Ultrasonography was then used to record the actual needle position and local anaesthetic spread. The anaesthetist performing the block was blinded to the ultrasound images.

Thirty six adult patients were included in this study,where in only seventeen patients the needle tip were found to be in transverse abdominis plane.They concluded that in standard blind transverse abdominis plane black was inaccurate, and the incidence of peritoneal placement is unacceptably high.

IN 2013, WALDRON et al [26] - Evaluated the impact of perioperative single dose systemic Dexamethasone for postoperative pain. Patients treated with Dexamethasone did not Demonstrate a significantly increased risk of infection or wound healing but blood glucose levels were higher at 24 hours

Fourteen studies (1449 patients) [1]examined the incidence of wound infection. From those, eleven found no reported infection related to the use of Dexamethasone or use of placebo in treated patients. In the three remaining studies (two-hundred thirty five patients), there was no increase of infection in patients receiving Dexamethasone.

Nine studies (1020 patients) examining the incidence of wound healing found no difference in either Dexamethasone-treated or placebo-treated patients. It has been found that a single perioperative dose of Dexamethasone, as opposed to long term use, has not been associated with delayed wound healing and increased risk of infection.

The proposed hypothesis for the mechanism of action as follows,

- Dexamethasone reduces stimulus **transmission in unmyelinated c-fibers**, known to carry nociceptive information by inhibiting the activity of the potassium channels on these fibers.

- It Causes a degree of **vasoconstriction** to the tissues and local anesthetic will have a slower uptake and absorption thus, prolonging its duration.

- It has Potent **anti-inflammatory effect** and inhibits the release of inflammatory mediators like interleukins and cytokines.
- It promotes the release of anti inflammatory mediators leading to **decreased postoperative pain**.

METHODS & MATERIALS

This study was conducted in the Obstetrics theatre, Kanyakumari government medical college after obtaining ethical committee approval and approval from Obstetrics & Gynecology department.

STUDY DESIGN

This was a Prospective Randomized double blinded study. This study was conducted in our department of Anaesthesiology after receiving ,

1.Institutional Ethical Committee approval

2.Department of obstetrics and gynecology approval and

3.Informed written consent from all the patients.

RANDOMISATION

Sample was randomized by closed envelope method.

60 plain covers each with a single sheet written B in 20 sheets, D1 in 20 sheets and D2 in 20 sheets were prepared and kept in the operation theatre.

- The covers were mixed thoroughly. Before the start of the surgery , theatre staff picked up a cover of his/her choice.
- If that cover contains a sheet written B in it, then the case was included in Bupivacaine Group (B) and vice versa.,
- This procedure were continued till all the 60 covers were exhausted , thereby enrolling 20 cases in each group.

SAMPLE SIZE

Sample size is calculated using the formula

$$n = \frac{2 \times \{z_{(1-a/2)} + z_{(1-b)}\}^2}{D^2}$$

The number of participants required in each intervention group was calculated as 14 for a Significance level of 99.9% and power of 95%.

Based on the figures for duration of anaesthesia by Amany S.Ammar et al in their study , “Effect of adding dexamethasone to bupivacaine on transversus abdominis plane block for abdominal hysterectomy: A prospective randomized controlled trial” published in Saudi Journal of Anaesthesia, July – Sept. 2012 6(3) : 229 -233.

- 60 Antenatal patients were studied.

GROUP ALLOCATION

Randomised into three groups –

GROUP (B, D1 and D2) - 20 patients each

- In group B – 20 ml of 0.25 % bupivacaine +2 ml of normal saline + 2 ml distilled water IV.
- In group D 1 – 20 ml of 0.25 % bupivacaine +2 ml (8 mg) dexamethasone + 2 ml distilled water IV.

- In group D 2 – 20 ml of 0.25 % bupivacaine +2 ml of normal saline + 2 ml (8 mg) dexamethasone IV.

BLINDING

The anaesthesiologist who administered the drug and the observer were blinded to the study. Local anaesthetic, study drug mixture was prepared by duty anaethesiologist not participating in the study. Postoperative observation was done by the same anaesthesiologist who administered the drug, who was unaware of the group allocation.

INCLUSION CRITERIA

- Age 20-35 years
- ASA II
- Posted for LSCS

EXCLUSION CRITERIA

- Patient refusal
- ASA grade III & IV
- Height <140 cms,BMI>30
- Known allergy to study drug
- Patient with coagulopathies
- Patient with diabetic, renal, liver diseases, pre eclampsia, eclampsia.

MATERIALS

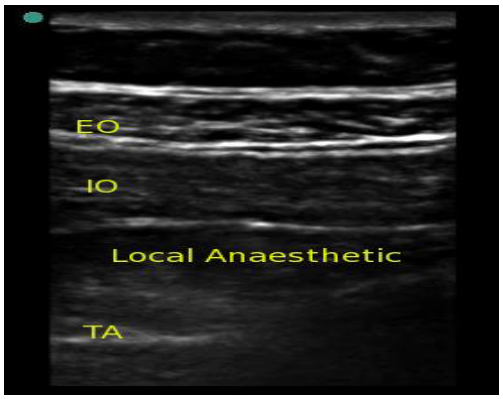
- Ultrasound machine with a high frequency probe (10-5 MHz)
- Ultrasound probe cover
- Antiseptic solution forskin disinfection
- Ultrasound gel
- 25 gauge quinke spinal needle
- 20ml needle and injection tubing
- Drug mixture for the block
- Equipments & drugs for resuscitation

INTERVENTION

All patients received premedication with Inj.Metoclopromide & Inj.Ranitidine intravenously before shifing the patient into operating room.Under strict aseptic precautions **Spinal Anaesthesia** was administered in Right Lateral position in L3 -L4 space using 10 mg 0.5% Bupivacaine using 25G Quinke's needle.

At the end of surgery,when the spinal anaesthesia level regressed to T8 , TAP block was given using **ultrasound guidance (sonoray)** by Curvilinear probe.The Transverse abdominis plane was approached through mid-axillary approach - **In Plane Technique** using a 23 Gauge quincke's spinal needle .

Drug was given and the correct spread of local anesthetic mixture was visualized through ultrasound.The same procedure was repeated on the other side of the abdomen.



ASSESSMENT OF BLOCK

After spinal regression below L2,checked for cold sensation at L1 dermatome.

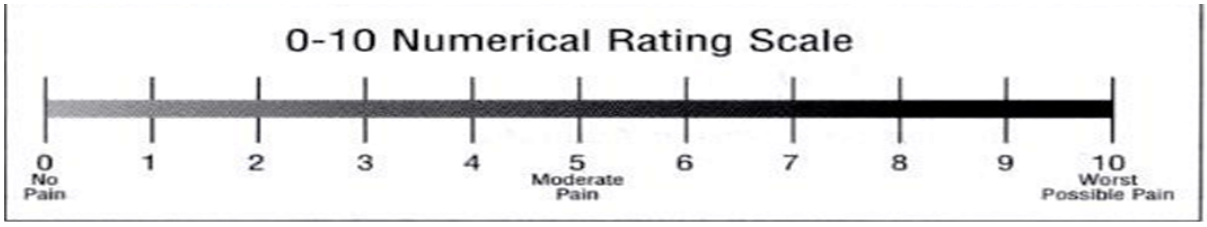
SUCCESS	Grade 2	Not able to perceive cold sensation at L1 on both sides .
PARTIAL	Grade 1	Able to perceive cold sensation at L1 on any one side .
FAILED	Grade 0	Able to perceive cold sensation at L1 on both sides .

OBSERVATION :

The following parameters were observed.

- Demographic data - Age, Weight.
- Block grade
- Numerical pain scale for 24 hrs postoperatively
- Duration of analgesia
- Total dosage of tramadol used
- Complications if any

The Numerical rating pain scale was compared at the time intervals 0,1,2,3,4,5,6,8,12,24 hrs during rest as well as movement



Inj. TRAMADOL 50 mg was given intramuscularly when Numerical rating scale score =/> 5 and the time was noted as time of first analgesic dose.

OUTCOME

PRIMARY OUTCOME - DURATION OF ANALGESIA

Time interval between the block time and the time of first analgesic dose.

COMPLICATIONS - Nausea, vomiting, transient femoral nerve palsy.

STATISTICAL TOOL

Data analysis was performed using IBM-SPSS version 20.0 (IBM-SPSS Science Inc., Chicago, IL).The above said datas were collected and tabulated.Data are presented as mean, standard deviation, percentages, or number of cases. Continuous data were compared by One way Anova. Significance was defined by P values less than 0.05. Non-parametric data were compared by Kruskal–Wallis one-way analysis of variance

OBSERVATION AND RESULTS

The datas are tabulated and analysed statistically using SPSS version 20.0

DISTRIBUTION OF AGE

	GROUP	MEAN	STANDARD DEVIATION	P VALUE
AGE	Group B	25.05	3.59	0.817
	Group D1	24.75	2.86	
	Group D2	24.25	5.23	

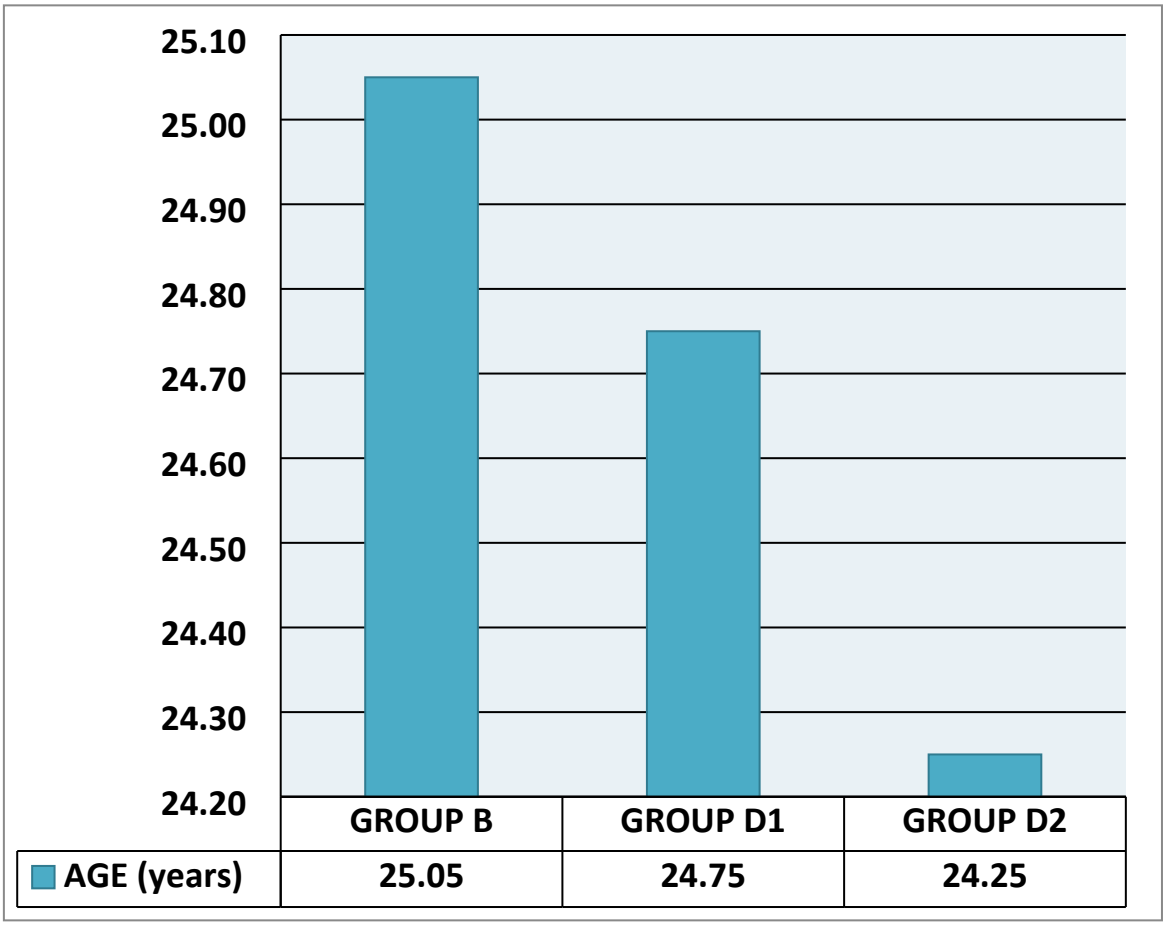
The age of all patients were comparable in all groups. Statistically analysed results for age were GROUP B 25.05 +/- 3.59 GROUP D1 24.75+/-2.86 and GROUP D2 24.25 +/- 5.23 and are not statistically significant with a p value 0.817.

DISTRIBUTION OF WEIGHT

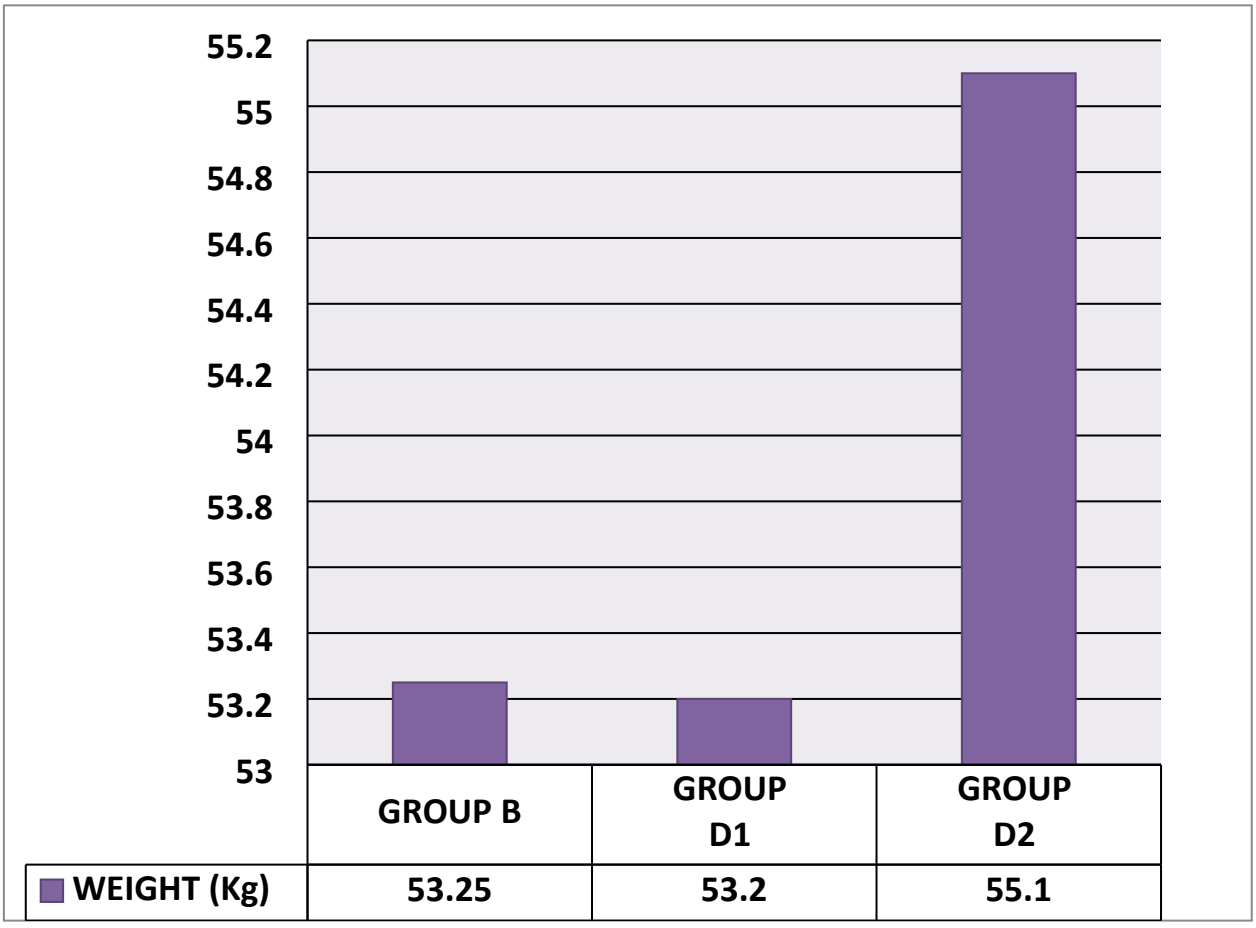
	GROUP	MEAN	STANDARD DEVIATION	P VALUE
WEIGHT	Group B	53.25	4.37	0.241
	Group D1	53.20	3.18	
	Group D2	55.10	4.35	

The statistically analysed results for weight were GROUP B 53.25 +/- 4.37, GROUP D1 53.2+/-3.18 and GROUP D2 55.1 +/- 4.35 which was not statistically significant with a p value 0.241.

AGE


25.10
25.00
24.90
24.80
24.70
24.60
24.50
24.40
24.30
24.20
GROUP B
GROUP D1
GROUP D2
AGE (years)
25.05
24.75
24.25

WEIGHT


55.2
55
54.8
54.6
54.4
54.2
54
53.8
53.6
53.4
53.2
53
GROUP B
GROUP D1
GROUP D2
WEIGHT (Kg)
53.25
53.2
55.1

BLOCK SUCCESS GRADE

GROUP	GRADE 2 SUCCESS	GRADE 1 PARTIAL	GRADE 0 FAIL
GROUP B	100 %	NIL	NIL
GROUP D1	100 %	NIL	NIL
GROUP D2	100 %	NIL	NIL

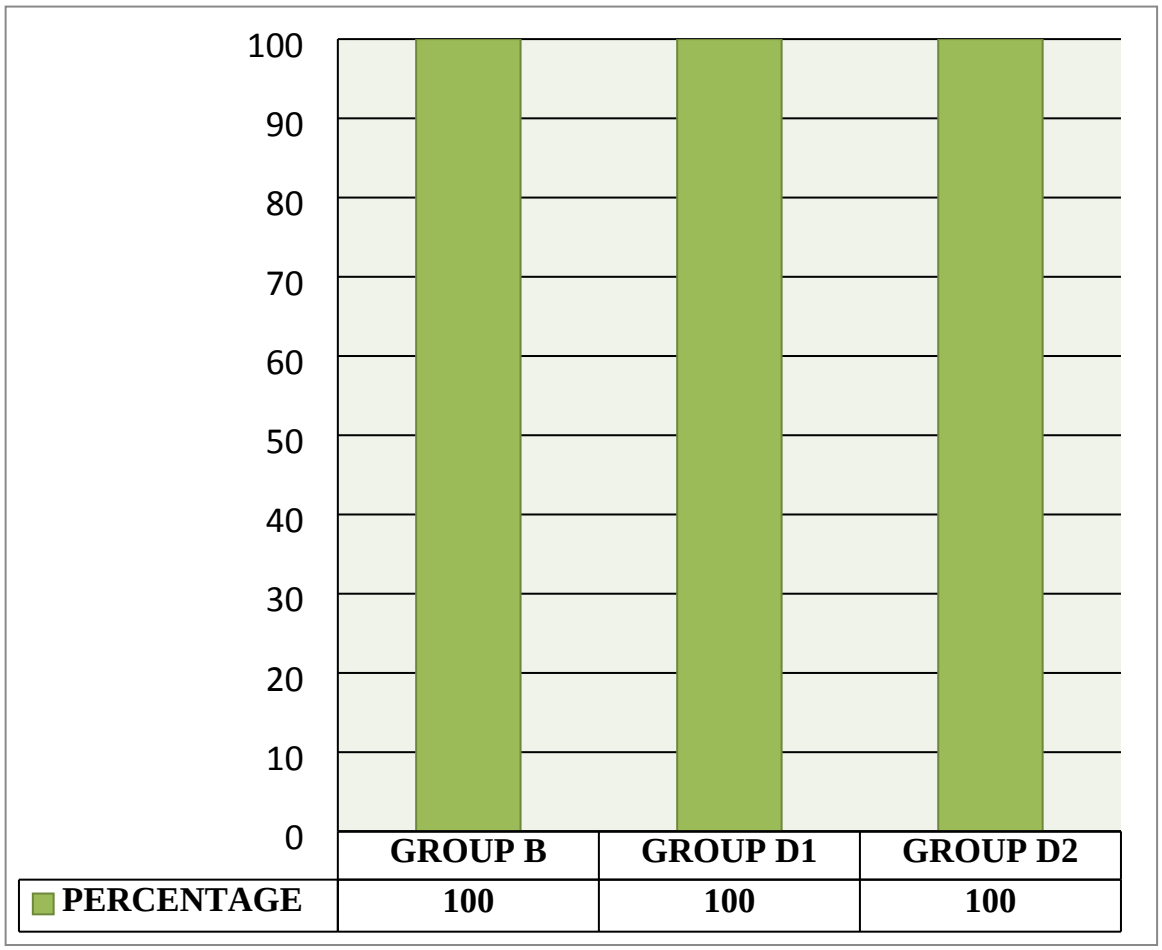
Transverse abdominis plane block were 100 % successful in all the three groups.

DURATION OF ANALGESIA

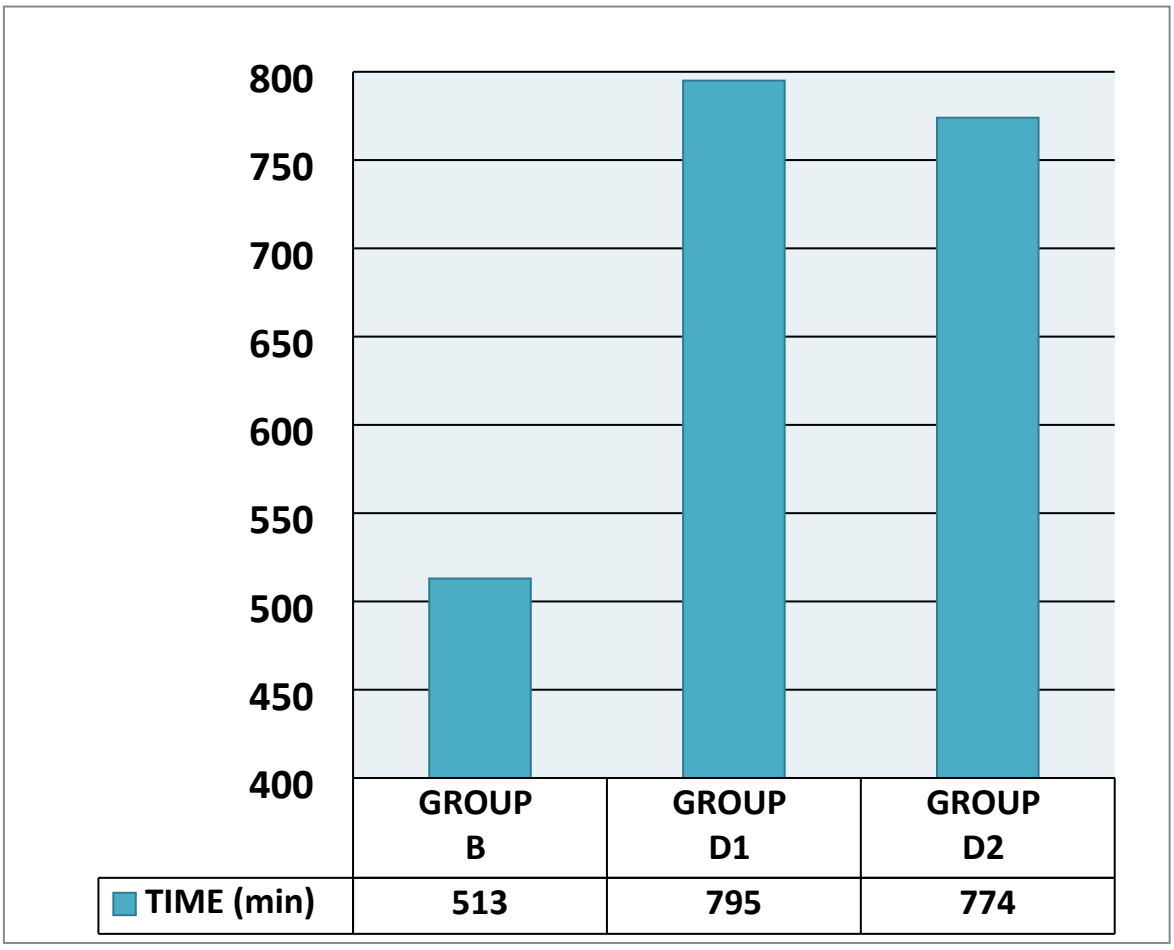
GROUP	MEAN	STANDARD DEVIATION	P VALUE
GROUP B	513.00	259.33	0.018 (significant)
GROUP D1	795.00	374.89	
GROUP D2	774.00	369.81	

The duration of analgesia in GROUP B was 513 minutes +/- 259.33 compared to 795 +/-374.89 minutes in GROUP D1 and GROUP D2 was 774 +/- 369.81 minutes with a significant p value of 0.018.

BLOCK SUCCESS GRADE



DURATION OF ANALGESIA



MULTIPLE COMPARISONS OF DURATION OF ANALGESIA

	COMPARISON BETWEEN GROUPS		MEAN DIFFERENCE	P VALUE
DURATION OF ANALGESIA	B	D1	-282	0.03
	B	D2	-261	0.04
	D1	B	282	0.03
	D1	D2	21	1.00
	D2	B	261	0.04
	D2	D1	-21	1.00

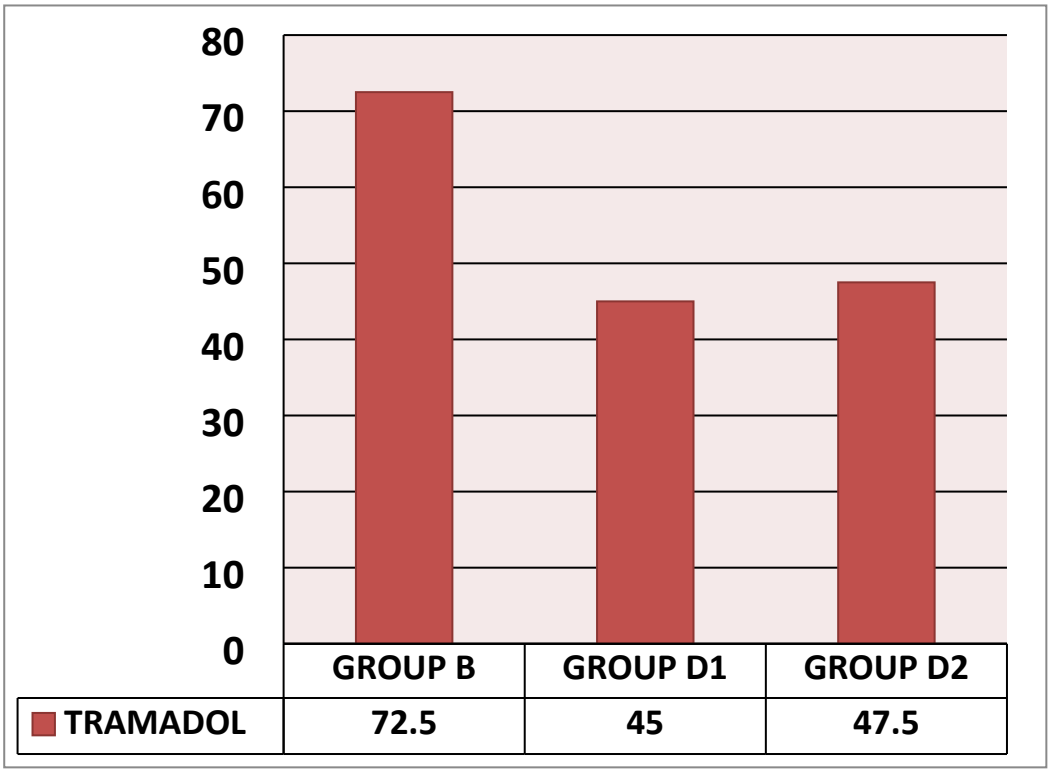
Comparison of duration of analgesia between three groups proved that the mean difference between GROUP B and GROUP D1 , GROUP B and GROUP D2 was statistically significant with a p value of <0.05.

TOTAL DOSE OF TRAMADOL USED

	MEAN (mg)	**STANDARD DEVIATION**	**P VALUE**
GROUP B	72.50	30.24	0.007 (significant)
GROUP D1	45.00	27.62	
GROUP D2	47.50	30.24	

The total dose of tramadol used were with a mean of 72.50 mg in GROUP B compared to 45 mg in GROUP D1 and 47.5 mg in GROUP D2 with a significant p value of 0.007.

TOTAL DOSE OF TRAMADOL USED



MULTIPLE COMPARISONS OF TOTAL DOSE OF TRAMADOL USED

	COMPARISON BETWEEN GROUPS		**MEAN DIFFERENCE**	**P VALUE**
TRAMADOL USED	B	D1	27.5	0.01
	B	D2	25	0.03
	D1	B	-27.5	0.01
	D1	D2	-2.5	1.00
	D2	B	-25	0.03
	D2	D1	2.5	1.00

Comparison of Total dose of tramadol used between three groups proved that the mean difference between GROUP B and GROUP D1 , GROUP B and GROUP D2 was statistically significant with a p value of <0.05.

COMPLICATION

None of the patients registered complications like nausea, vomiting, dizziness, femoral nerve palsy, pelvic hematoma, liver trauma etc. during first 24 hour study period.

DISCUSSION

Several researches have revealed that addition of Dexamethasone to local anaesthetics proved to prolong the duration of analgesia and reduces rescue analgesic requirements. Several mechanism have been hypothesized to explain that addition of Dexamethasone reduces pain transmission in C fibres, vasoconstriction ,and also has anti inflammatory effect.This study is designed to find out wheather there is any difference in action of Dexamethasone given via perineurally or systemically when added to local anaesthetics.

Abdallah et al in 2015[15] conducted a study comparing Intravenous dexamethasone and perineural dexamethasone for supraclavicular brachial plexus block using 30-mL bupivacaine 0.5% alone (Control), with concomitant intravenous dexamethasone 8 mg , or with perineural dexamethasone 8 mg . The duration of analgesia was prolonged in the intravenous Dexamethasone group (25 hours) and perineural Dexamethasone group (25 hours) when compared to Control group (13.2 hours) with a p value of < 0.001 .

Consistent with this study , in my study also the duration of analgesia is prolonged in perineural Dexamethasone group (795 +/- 374.89 min) when compared to Bupivacaine group (513 +/-259.33 min) with a p value of 0.03 and also duration of analgesia is prolonged in intravenous Dexamethasone group (774 +/- 369.81 min) when compared to Bupivacaine group with a p value of 0.04 with no statistical significant difference between perineural and intravenous group (p 1.00) .

Similar results have been obtained cby Desmet et al in 2013 who Performed a study on intravenous Dexamethasone and its equivalency to perineural Dexamethasone in prolonging the analgesic duration of single-shot interscalene block with Ropivacaine where the median time of a sensory block was equivalent for perineural and i.v. dexamethasone: 1405 min and 1275 min respectively. There was a significant difference between the Ropivacaine group (757 min) and the Dexamethasone groups (P<0.0001) and Rahangdale et al in 2014[23]who conducted a study to assess the effects of perineural versus intravenous dexamethasone on sciatic nerve blockade by using 0.5% bupivacaine with epinephrine 1:300,000 (0.45 mL/kg) and he concluded that there is no significant difference in the time to first toe movement or analgesic duration between the perineural (P < 0.001) and IV dexamethasone groups (P = 0.008) .

Conflicting results have been reported by KAWANISHI et al in 2014 [16]conducted a study to compare perineural Dexamethasone vs systemic low-dose dexamethasone 4mg in interscalene block with Ropivacaine 0.75% , The median times of sensory block in Ropivacaine group was 11.2 hours , in perineural dexamethasone group was 18.0 hours, and in intravenous dexamethasone group was 14.0 hours and he concluded that Perineural but not intravenous administration of 4 mg of dexamethasone significantly prolongs the duration of postoperative analgesia.

In my study, total dose of rescue tramadol used is higher in Bupivacaine group (72.50 +/- 30.24 mg) when compared to perineural Dexamethasone group (45 +/- 27.62 mg) in first 24 hours which is statistically significant with a p value of 0.01 and similarly total rescue tramadol dose used is lower in intravenous Dexamethasone group (47.50 +/-30.24 mg) compared to plain bupivacaine group with a p value of 0.03.This observation correlates well with the study conducted by Deshpande et al in 2017 [22]to check the Analgesic Efficacy of Dexamethasone Added to Ropivacaine in Transversus Abdominis Plane Block for Transabdominal Hysterectomy under Subarachnoid Block and concluded that lesser Tramadol requirement in first 24 h in Ropivacaine dexamethasone group (50.2 ± 34 vs. 94 ± 35 mg, $P < 0.001$) as compared to Ropivacaine group.

No studies were available to compare the rescue analgesic requirement between intravenous and perineural dexamethasone goup but in my study there is no significant difference in the requirement of tramadol between perineural and intravenous group with a p value 1.00.

No complication such as nausea, vomiting, dizziness, femoral nerve palsy, pelvic hematoma, liver trauma were recorded during this study period.

SUMMARY

To summarise, All the demographic data like age and weight are comparable. In this study either Perineurally or Intravenously added Dexamethasone to Bupivacaine in Transverse abdominis plane block prolongs the duration of analgesia (795 min Vs 774 minutes) as compared to plain bupivacaine group (513 min).The rescue analgesic tramadol dose is reduced in both groups GROUP D1 and D2 (45 Vs 47.5 mg) as compared to plain Bupivacaine group 72.5 mg .No complication was reported.

CONCLUSION

The Dexamethasone in the dose of 8 mg added either Perineurally or Intravenously to 0.25 % Bupivacaine in Transverse abdominis plane block in cesaerean patients prolongs the duration of the block significantly and also reduces postoperative rescue analgesic requirement in the first 24 hours compared to plain Bupivacaine group .

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PROFORMA

Date -

Name - Age/Sex- IP.NO - ASA Risk- weight -

Diagnosis- Type Of Surgery- Under Spinal Anaesthesia-

PRE ANAESTHETIC STATUS-

Pulse Rate- Blood Pressure- Cvs- Rs-

BLOCK	SUCCESS	PARTIAL	FAIL

SPINAL ANAESTHESIA	PERFORMANCE OF BLOCK	L 2 REGRESSION	FIRST ANALGESIA	DURATION OF ANALGESIA

TIME	NUMERICAL RATING SCALE		DOSE OF RESCUE ANALGESIC	COMPLICATION
	REST	MOVEMENT		
0 hr				
1hr				
2 hr				
3 hr				
4 hr				
5 hr				
6 hr				
8 hr				
12 hr				
24 hr				

MASTER CHART

GROUP B

	Age	Weight	IP no	Block grade	Time of SAB	Performance of Block	L2 regression	First rescue analgesia	Duration of analgesia (min)	NRS0		NRS1		NRS2		NRS3		NRS4		NRS5		NRS6		NRS8		NRS12		NRS24		Total Tramadol (mg)	Complications
										R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M		
1	29	50	20096	2	6pm	8pm	10.30pm	4am	480	0	0	1	1	2	2	2	2	3	3	3	3	3	3	6	6	3	3	3	3	50	nil
2	25	50	20179	2	3pm	4pm	7.30pm	4am	720	0	0	1	1	2	2	3	3	3	3	4	4	4	4	4	4	4	4	7	7	50	nil
3	25	60	19325	2	6.30pm	7.45pm	9pm	3.45am	480	4	4	4	4	3	4	4	2	2	2	3	3	2	2	7	7	3	3	7	7	100	nil
4	24	55	19606	2	10.30pm	11.15pm	12.30am	7.15am	480	0	0	1	1	4	4	3	3	3	3	3	3	4	4	6	6	7	7	3	3	100	nil
5	20	50	19654	2	10.45pm	11.30pm	1.20pm	7.30pm	480	0	0	1	1	2	2	3	3	3	3	4	4	4	4	6	6	7	7	3	3	100	nil
6	30	45	26004	2	6.45pm	8pm	8.30pm	10pm	120	0	0	0	0	7	7	2	2	2	2	2	2	3	3	3	3	3	3	6	6	100	nil
7	22	50	26020	2	6.30pm	7.45pm	8pm	3.45am	480	0	0	2	2	4	4	7	7	3	3	3	3	3	3	6	6	4	4	6	6	100	nil
8	22	57	18821	2	4.30pm	5.30pm	6.30pm	9.30pm	480	0	0	0	0	3	3	3	3	4	4	4	4	4	4	6	7	3	3	7	7	100	nil
9	23	50	25219	2	6.30pm	8.45pm	10pm	10.45pm	120	1	1	2	4	5	6	2	2	2	2	2	2	2	2	2	2	3	3	6	6	50	nil
10	30	55	26112	2	10.30am	11.15am	12.30pm	7.15pm	480	0	0	1	1	1	1	2	2	2	2	2	2	4	4	7	7	3	3	3	3	50	nil
11	28	55	26267	2	3pm	4.30pm	6pm	4.30am	1440	0	0	2	2	2	2	2	2	2	2	2	2	2	2	4	4	4	4	7	7	0	nil
12	31	55	26223	2	6.30pm	9pm	10.30pm	5am	480	0	0	2	2	2	2	3	3	3	3	2	2	3	3	6	6	3	3	4	4	50	nil
13	23	50	25929	2	10.45am	11.45am	1pm	7.45pm	420	0	0	2	2	2	2	2	2	3	3	3	3	4	4	7	7	3	3	7	7	100	nil
14	23	56	25419	2	10.30am	11.45am	1pm	7.45pm	480	0	0	1	1	1	1	1	1	2	2	2	2	3	3	6	6	3	3	3	3	50	nil
15	20	55	26249	2	3pm	4.45pm	6pm	12.45am	480	0	0	2	2	2	2	3	3	3	3	3	3	3	3	7	7	3	3	6	6	100	nil
16	24	45	26345	2	7am	8.30am	10am	4.30pm	480	0	0	2	2	2	2	3	3	3	3	3	3	3	3	7	7	3	3	2	2	50	nil
17	26	53	26789	2	5.30am	7.30am	9.45am	7.30pm	720	0	0	0	0	1	2	3	3	3	3	3	4	3	3	3	3	7	7	6	7	100	nil
18	20	55	27432	2	6am	8am	10am	4pm	480	0	0	2	2	2	2	3	3	3	3	3	3	4	4	6	6	3	3	4	4	50	nil
19	30	59	27985	2	7pm	8.15pm	10pm	4.15am	480	0	0	2	2	2	2	3	3	3	3	3	3	3	3	7	7	3	3	7	7	100	nil
20	26	60	28012	2	10pm	11.30pm	12.54am	7.30am	480	0	0	2	2	2	2	3	3	3	3	3	3	4	3	7	7	3	3	3	3	50	nil

GROUP D1

S. No	Age	Weight	IP no	Block grade	Time of SAB	Performance of Block	L2 regression	First rescue analgesia	Duration of analgesia (min)	NRS0		NRS1		NRS2		NRS3		NRS4		NRS5		NRS6		NRS8		NRS12		NRS24		Total Tramadol (mg)	Complications
										R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M	R	M		
1	21	58	20116	2	9.45am	11.20am	12pm	2.20pm	180	0	0	0	0	3	3	5	5	1	1	2	2	3	3	4	4	4	4	4	4	50	nil
2	22	55	19737	2	9.15pm	10.40pm	11.50pm	12.40pm	720	0	0	1	1	1	1	2	2	3	3	3	3	3	3	3	3	8	8	4	4	50	nil
3	22	55	19287	2	8pm	9.15pm	10pm	11.15pm	120	2	2	3	3	6	6	4	4	4	4	3	3	4	4	3	4	4	4	3	3	50	nil
4	28	55	17974	2	5pm	6.30pm	7.30pm	6.30pm	1440	0	0	1	1	1	1	2	2	2	2	3	3	3	3	3	3	4	4	4	4	0	nil
5	25	48	18904	2	9.15pm	10.30pm	11.30pm	12.30pm	720	3	3	4	4	3	3	4	4	4	4	4	4	3	4	4	4	6	6	4	4	50	nil
6	24	55	19264	2	9.30pm	10.30pm	12am	12.30pm	720	3	3	3	3	4	4	4	4	4	4	4	4	4	4	4	4	6	6	4	4	50	nil
7	28	48	19244	2	9.45pm	10.45pm	1am	12.45pm	1440	0	0	2	2	3	3	3	3	3	3	4	4	4	4	4	4	4	4	4	4	0	nil
8	23	52	19146	2	9pm	10pm	11.30pm	12pm	720	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	6	6	4	4	50	nil
9	22	58	19446	2	9pm	10pm	11.15pm	6am	480	1	1	1	1	1	1	1	1	2	2	2	2	2	2	6	6	3	3	7	7	100	nil
10	28	56	20572	2	7pm	8.10pm	9.30pm	10.10am	720	0	0	1	1	1	1	2	2	2	2	2	2	3	3	4	4	7	7	3	3	50	nil
11	24	55	11720	2	3.30pm	4.30pm	6.30pm	4.30pm	1440	0	0	0	0	0	0	2	2	2	2	2	2	3	3	3	3	4	4	7	7	50	nil
12	27	54	25323	2	7pm	9pm	10pm	5am	720	1	1	3	4	2	2	2	2	2	2	3	3	3	3	3	3	6	6	3	3	50	nil
13	30	52	26220	2	5.45pm	7.45pm	8.15pm	9.45pm	720	0	0	0	0	2	2	2	2	2	2	2	2	2	2	3	3	6	6	4	4	100	nil
14	23	56	26457	2	4.30pm	5.45pm	7.45pm	5.45am	720	0	0	0	0	2	2	2	2	2	2	2	2	3	3	3	3	6	7	3	3	50	nil
15	25	54	26832	2	5am	7am	8am	7pm	720	0	0	0	0	2	2	2	2	2	2	2	2	3	3	3	3	7	7	3	3	50	nil
16	28	54	27654	2	6.30am	8am	9.45am	8pm	720	0	0	1	1	2	2	2	2	2	2	2	2	3	3	3	3	3	3	3	3	0	nil
17	22	52	26976	2	5pm	7pm	8.30pm	7am	720	0	0	1	2	2	2	2	2	2	2	2	2	3	3	3	3	6	7	4	4	50	nil
18	26	49	27012	2	7pm	9pm	10pm	9am	1440	0	0	2	2	2	2	2	2	2	2	2	2	3	3	3	3	3	3	4	4	0	nil
19	27	48	26544	2	10pm	11.30pm	12am	11.30am	720	0	0	2	2	2	2	2	2	2	2	3	3	3	3	4	4	7	7	3	3	50	nil
20	20	50	28766	2	10am	11.45am	12.30pm	11.45pm	720	0	0	1	2	2	2	2	3	2	2	2	3	3	3	3	3	7	7	3	3	50	nil

GROUP D2

S. No	Age	Weight	IP no	Block grade	Time of SAB	Performance of Block	L2 regression	First rescue analgesia	Duration of analgesia (min)	NRS0		NRS1		NRS2		NRS3		NRS4		NRS5		NRS6		NRS8		NRS12		NRS24		Total Tramadol (mg)	Complications
41	38	60	8673	2	12.10pm	1.45pm	3pm	1.45am	720	0	0	0	0	1	1	1	2	2	2	2	3	3	3	4	4	5	6	4	4	50	nil
42	21	55	8703	2	6.20 pm	8:00 PM	9.30pm	8:00 AM	720	0	1	1	2	2	3	2	3	3	4	3	4	4	4	4	4	5	7	4	4	50	nil
43	19	50	8633	2	4.20 pm	6:00 PM	7:00 PM	6:00 PM	1440	0	1	0	1	1	2	1	2	2	3	2	3	3	3	4	4	4	4	4	4	0	nil
44	28	58	9583	2	10pm	11.30 pm	12.45 pm	11.30am	720	0	1	0	1	1	2	1	3	2	3	3	3	3	4	3	4	5	6	4	4	50	nil
45	21	55	9636	2	9.40pm	10.50pm	12am	9.50am	720	0	0	1	1	1	2	2	3	2	3	3	3	4	4	4	4	5	6	4	4	50	nil
46	24	60	9922	2	11.45pm	1am	2am	9am	480	0	0	1	2	2	3	2	3	3	3	3	3	4	4	6	6	4	4	4	4	50	nil
47	22	55	10067	2	4.50pm	7pm	7.30pm	7am	720	0	0	1	1	2	2	2	3	3	4	3	4	4	4	4	4	6	7	3	3	50	nil
48	23	60	10066	2	4.30pm	6.30pm	7pm	6.30 am	720	0	0	1	1	1	2	2	3	2	3	2	3	4	4	4	4	7	7	3	3	50	nil
49	20	60	10199	2	5.15pm	6.45pm	8pm	6.45am	720	0	1	0	2	1	2	1	2	2	3	2	3	3	3	3	3	5	7	4	4	50	nil
50	14	50	12703	2	6pm	7pm	8.30pm	11pm	240	0	0	0	1	3	2	3	3	5	6	3	3	3	3	3	3	3	3	4	4	50	nil
51	30	55	10890	2	5.30pm	7pm	8pm	7am	720	0	1	0	2	1	2	1	3	2	3	2	3	3	3	3	4	6	6	4	4	50	nil
52	29	60	10810	2	6pm	7.30pm	8.30pm	0	1440	0	0	1	1	1	2	2	3	2	3	3	3	3	3	3	3	3	3	3	3	0	nil
53	31	60	10650	2	10pm	11.30pm	12.30pm	6.30am	420	0	0	0	0	1	2	2	3	2	3	3	3	6	7	2	3	3	3	6	7	100	nil
54	23	50	12084	2	9.30pm	11pm	12.30am	5am	420	0	1	2	3	3	3	3	4	4	4	4	5	6	7	3	3	6	6	3	3	100	nil
55	27	50	12157	2	5.30pm	7pm	8.30pm	7am	720	0	1	1	2	1	2	2	3	2	3	3	4	4	5	4	5	6	6	3	4	50	nil
56	24	50	12080	2	10.15pm	11.45pm	1am	7.45am	480	0	1	0	1	1	2	1	2	2	3	2	3	3	4	6	7	3	3	7	7	100	nil
57	26	50	11076	2	11.30am	1pm	2pm	9pm	480	0	1	1	1	1	2	2	2	2	3	3	3	4	3	6	7	3	3	3	3	50	nil
58	23	60	11276	2	12pm	2.30pm	3.30pm	2.30am	720	0	0	0	1	1	1	2	2	2	3	3	3	4	4	4	4	6	7	3	3	50	nil
59	20	50	12532	2	1pm	2.45pm	4pm	0	1440	0	0	0	0	1	1	1	2	2	2	3	3	3	3	3	3	3	4	3	4	0	nil
60	22	54	11234	2	4pm	6pm	8pm	0	1440	0	1	1	1	1	1	2	2	2	3	3	3	3	3	3	3	3	4	4	4	0	nil